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^cDISEASES OF THE STOMACH

INCLUDING

DIETETIC AND MEDICINAL TREATMENT

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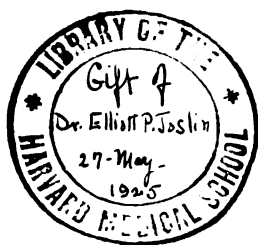
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ILLUSTRATED WITH 126 ENGRAVINGS AND 15 PLATES



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1913



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PREFACE

It has been the author's intention to describe the diseases of the stomach as he has happened to see them, and to present these subjects from the standpoint of personal experience. For this purpose series of cases have been grouped and analyzed and the results noted, whether or not these results have been in harmony with preëxisting ideas. When the results have been at variance with the accepted teachings, the fact has been noted, and opposing views given free discussion, but no attempt has been made to alter the results of the analyses of the case histories because they may seem strange and unexpected. Each series has comprised as many cases as possible, differing naturally in number according to the frequency of the disease under discussion. In the analyses of diseases of comparative rarity, the recorded cases have, as a rule, been read in full in the original articles and the statistics thus carefully worked out. The following pages, therefore, do not represent a compilation from various authorities, brought together and harmonized, but, on the contrary, they reflect careful study of conditions of disease from private and hospital case records and from authentic histories in literature.

The book may be described, in brief, as a fairly complete presentation of diseases of the stomach as the result of many years of practice and observation of this special field. It is hoped that the general practitioner may find in it what he needs, and that the specialist may be interested in the author's views when they differ from those commonly accepted hitherto.

To many friends who have helped and encouraged the author in this task he is deeply grateful. To Dr. Albert R. Lamb, of the Presbyterian Hospital, he is indebted for invaluable assistance in the sections on Pathology and for the supervision of the photographs of pathological material. His thanks are tendered to Dr. Robert L. Hutton for valuable assistance in the preparation of many of the articles. To Dr. Hoobler he is under obligations for his assistance in the preparation of the article on Cyclic Vomiting. The sections on Radiographic Diagnosis have been difficult to prepare, owing to radical differences of opinion between some of the leading radiologists as to the limita-

tions, or lack of limitations, of their special art. Many minor points in radiographic diagnosis have not been given in the text simply because of differences at the present time between these experts as to their diagnostic value. To Dr. Busby, of the New York Hospital, and Dr. Le Wald, of St. Luke's Hospital, the author desires to express his thanks for their kindness in supplying illustrative radiographic plates. Acknowledgment of the donor of each plate is made in the text. Dr. Edward Leaming has not only furnished many of the plates, which are severally acknowledged in the text, but also has reproduced the plates from all sources, so as to be suitable for illustration, and has, furthermore, by his helpful criticism, been of the greatest personal service. To him grateful thanks are rendered. To Dr. Bloodgood, of Baltimore, the author is indebted for a number of photographs of pathological specimens from his laboratory at Johns Hopkins Hospital. To other friends, and to those collaborators who have kindly allowed the use of illustrations from their articles, an expression of appreciation of their courtesy is due. Acknowledgment of the source of each illustration is given in the text.

G. R. L.

NEW YORK, 1913.

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DISEASES OF THE STOMACH

CHAPTER I

ACUTE GASTRITIS

ACUTE CATARRHAL GASTRITIS

ALTHOUGH acute gastritis is undoubtedly one of the commonest of diseases, the number of cases actually coming under the observation of the physician is relatively small, as in the great majority of instances the symptoms are either transient or of such mild severity that no medical treatment is required. Unfortunately, however, the diagnosis of acute catarrh of the stomach is made far more frequently than is warranted, and often includes cases of acute appendicitis, gall-bladder disease, ulcer, acute onset of cancer of the stomach, neuroses, and even the gastric crises of tabes. The term, strictly speaking, should only be applied to those cases of acute indigestion in which direct evidence of gastric inflammation exists, and not indiscriminately used to include all temporary subjective disturbances that follow dietetic errors. It is, however, extremely difficult to determine, clinically, whether or not a true inflammation exists, as pathognomonic signs of such are usually lacking, except in the infectious forms that are the result of food poisoning.

Acute catarrhal gastritis may be clinically divided into two prominent types:

1. Dietetic or simple gastritis.
2. Infectious gastritis or food poisoning.

I. Dietetic or Simple Gastritis.—Etiology.—Among the causes predisposing to acute catarrhal gastritis may be enumerated lesions of the heart or of the liver, leading to chronic congestion of the stomach. Conditions of depreciated health or of fever that render it difficult for the stomach properly to digest its food, may be considered as contributory causes, although the influence of the general condition of health is far less potent than is ordinarily supposed. It is said that gouty individuals are liable to recurrent attacks of gastric catarrh.

The resistance of the stomach to inflammation differs very much in different people—a fact which we recognize by the use of such terms as “strong stomachs” and “weak digestions.” There are those whose stomachs, while equal to the ordinary daily task of digestion, are not possessed of any reserve power, but succumb to dietetic errors to which others seem immune.

There are, moreover, individual idiosyncrasies in which some normal and healthy food produces acute gastric irritation, that cannot be ascribed to toxic effects or to bacterial invasion. Thus, some persons suffer from vomiting and urticaria from eating fish in any form, or crabs or lobsters, while in others, beef, pork, and game will produce the same unpleasant results. It is not to be denied, however, that many of these instances are really cases of autosuggestion, from subconscious impressions derived from some previous unpleasant experience with that particular article of food. It is probable that many of these so-called idiosyncrasies may be explained by the recent theory of anaphylaxis.

During the earlier years of life the chief cause is found in dietetic errors. In old age the tendency to portal stasis renders the ailment relatively common. In these aged subjects the effect of exposure to cold may be sufficient to induce an attack, by causing a sudden increase in the internal congestion. Among other predisposing causes in elderly persons may be mentioned the frequency with which achylia is found in those aged over sixty years. The effect of such deficiency of gastric digestion in inducing an attack of acute gastric catarrh can readily be appreciated.

A predisposition to gastric catarrh is present during infectious disease, intestinal parasites, acute renal or cardiac disease, and often accompanies acute tuberculosis, masking the primary disease in many cases in which the pulmonary symptoms and physical signs are not evident.

Infection of gastric mucous membrane as the result of bacterial invasion is common enough. Gastritis due to streptococcus, typhoid bacillus, or pneumococcus is generally well recognized. These bacteria usually obtain entrance by the blood or lymph channels, and the gastritis is a secondary manifestation of the original infective disease. Pneumococcus infection may occur as a primary form of invasion, and may give rise to gastric ulceration.

Exciting Cause.—The commonest exciting cause for acute gastritis is dietetic error. This may occur in a great variety of ways.

1. *Errors in Food.*—The food may be too rich, or may be improperly cooked. Overeating is one of the most frequent causes. Drinking of cold liquids or of ice-cream soda when overheated is a common cause

for acute gastric catarrh, as is also overindulgence in alcohol. Attacks in children commonly follow the eating of unripe fruit. The influence of idiosyncrasy in diet has already been alluded to.

2. *Errors in Eating.*—Food may be proper and wholesome, but if imperfectly masticated, or bolted, or eaten when the patient is over-tired or under nervous strain, gastric catarrh may ensue.

The form of gastritis that follows the ingestion of infected food is described under a separate heading.

Swallowed secretions from the nasopharynx or bronchi may act as infective agents, especially potent in this regard, being decomposed pus from gangrene of the lung and the alveolar secretions of Rigg's disease.

Pathology.—No one has had a better opportunity of studying the gross appearance of the stomach in this condition than William Beaumont in the course of his prolonged experiments on Alexis St. Martin. He saw the condition as it existed in the living subject without the postmortem changes which render a correct appreciation of the true appearance so difficult. Morbid changes, following errors in eating and drinking, presented themselves several times in St. Martin's stomach during Beaumont's investigations. He thus describes an attack of acute gastritis:

"The coats of the stomach have not appeared in their usual healthy condition for several days past—the color darker; mucous coat unequal, some patches of a purplish color with aphthous edges, surface inclined to be dry, very little secretion of gastric juice, digestion slower and less perfect than usual, bowels inactive."

In another place, under similar conditions, he speaks of the "large proportion of thick, ropy mucus" mixed with the stomach contents.

This practically represents our conception of the condition at the present time. The stomach is not altered in size or shape. The changes are most marked at the pylorus, the region which bears the brunt of the digestive process. There is diffuse or patchy redness, with swelling of the mucous membrane. At times, there may be minute hemorrhages or superficial erosions. An excessive amount of tenacious mucus, occasionally of brownish tinge from extravasation of blood, covers the surface. The amount of gastric juice secreted is much less than normal, and it contains a diminished amount of hydrochloric acid.

Microscopically the changes are confined almost entirely to the mucosa. The glandular epithelium is swollen and granular, and it is difficult to distinguish between the chief and parietal cells. Goblet cells are numerous. The capillaries and small vessels are dilated and there may be many red blood cells scattered through the mucosa, with

some round-celled infiltration between the tubules and extending into the submucosa, which is often edematous. The solitary lymph follicles are often swollen, and leukocytes may be present in the intertubular tissue. The lumina of the glands contain debris of degenerated cells. Leukocytes, red blood cells, and desquamated epithelial cells, in various stages of degeneration, are mixed with the stringy mucus covering the surface.

Symptoms.—The symptoms begin usually within a few hours after the exciting cause, but may be delayed for twenty-four to thirty-six hours.

In the mild cases, nausea and vomiting may be absent, the chief complaints being headache, mental depression, loss of appetite and coated tongue. The bowels may be constipated or there may be diarrhea. An example of such an attack is as follows:

Male, aged forty-two years.

One week ago he went to a country hotel, where he ate heartily of badly cooked food. Next morning he awoke with a headache, coated tongue, and complete anorexia. He felt depressed and miserable. He remained in bed two days on a milk diet, and gradually improved, although his appetite was a long time in returning. His bowels, though previously regular, became constipated and moved only after taking calomel.

In the *more severe cases* the patient complains of uneasy feelings in the stomach that merge into nausea. Vomiting soon occurs, the vomited matters consisting of the contents of the stomach, mixed with mucus, and usually offensive from the presence of organic acids. There is almost invariably a reduction or even an absence of hydrochloric acid, although very rarely this form of acidity may be normal or excessive. Acute gastritis, with a total acidity of 40 or over, should arouse our suspicions that more than simple acute catarrh is present. Lactic acid is rarely present.

The tongue is usually dry and coated, and there is apt to be thirst. A diffuse tenderness may be elicited by pressure on the epigastrium.

In ordinary gastritis the *vomiting usually ceases as soon as the stomach is emptied, and is repeated only after ingestion of food*. When the vomiting is prolonged, the diagnosis of gastritis should not be made without reservation.

Watery vomiting or the vomiting of large quantities of fluid containing free hydrochloric acid does not occur in acute gastritis, but suggests some form of pyloric closure, commonly seen with ulcer, more rarely with cancer of the stomach.

Pain as a symptom of acute gastritis is not usually observed except in the attacks that follow a gross dietetic error. In these cases cramp-

like pains in the epigastrium may occur, later becoming more generally distributed over the lower abdominal zones, as the undigested ingesta enter the intestinal tract. This is a true "stomach-ache" common in childhood. The following case may be given as an example:

S. G., male, aged twenty-one years. Admitted May 8, 1909.

Present History.—Always well. On the day of admission he started with friends to "enjoy a day in the city." After visiting several saloons and eating immoderately of pickles and cabbage, he began to feel nauseated and to vomit frequently. He had cramp-like pains in the epigastrium.

Physical Examination.—Tenderness in the epigastrium was present with slight rigidity over the upper right rectus. Otherwise negative. He remained in the hospital for three days and was then discharged cured.

Pain other than this does not appear as a symptom of acute gastritis, despite the fact that many writers mention pain of a burning or lancinating character as not uncommon.

Flatulence does not occur unless it be a preëxisting symptom. The urine is usually scanty, highly colored, and cloudy from the precipitation of urates.

In mild cases the temperature is not usually elevated, although predisposition to fever is often peculiar to the individual, and the tendency to develop fever from slight causes may last through adult life. The temperature may be slightly elevated in the more severe cases.

In children, fever may be a prominent symptom and may be continuous for a number of days. To this condition, Eustace Smith has applied the term "acute febrile gastritis of children."

Prostration and headache are common, but are not, as a rule, severe except with food infections. There may be various forms of skin eruptions, chiefly of a roseolar, erythematous, or urticarial character, but these are rare, except after food poisoning, especially by shell-fish.

Diarrhea often appears at the onset, and may be due either to increased intestinal peristalsis or to a complicating ileocolitis. When the stomach alone is involved, constipation is the rule.

As the attack subsides, symptoms of atony often appear, and may protract the course of the disease several days or even weeks. There is lack of appetite or even nausea, though vomiting is rare. A slight amount of gastric flatulence may be present in this atonic stage. These symptoms are transient, however, and no sequelæ result.

Duration.—The duration of the attack is short, the acute stage rarely extending over one or two days, although it may be a week or so before the patient regains his appetite and considers himself well.

Cases with nausea and vomiting extending over two days must be regarded with suspicion. Recurrences are not common, except in those whose life history shows a constant repetition of attacks from individual predisposition, or in those who repeatedly subject themselves to gross dietetic errors. *Frequent recurrences without assignable cause should throw doubt upon the diagnosis.*

Diagnosis.—The extreme frequency in which acute appendicitis first manifests itself by pain or distress in the epigastrium and vomiting should always be remembered, and a careful examination should be made, even in cases that seem straightforward examples of acute gastric catarrh. The blood count is of value in doubtful cases.

Exacerbations of chronic appendicitis may be responsible for many cases of apparent gastritis, with pain, nausea, and vomiting, often associated with diarrhea. This is especially the case in children in whom recurring bilious attacks with nausea and vomiting often cease after a frank attack of inflammation of the appendix, and its subsequent removal.

Ulcer of the stomach is often erroneously diagnosed as gastritis, especially when the former disease assumes the vomiting type. The vomiting of ulcer is, however, more protracted than that of gastritis, is associated with definite pain, and assumes in many instances the acid watery type of vomiting which is never seen in gastric catarrh. Many cases of cancer begin with vomiting, and it is suspicious of malignancy if, during the age of cancer incidence, protracted vomiting occurs without an apparent exciting cause, especially if the patient has recently been losing somewhat in flesh and strength.

The gastric symptoms of gall-bladder disease are often erroneously diagnosed as gastritis. Physical examination shows the tenderness located over the gall-bladder area, which should prevent error in diagnosis between these two conditions. It must be remembered that many of the acute infectious diseases may begin with vomiting, as an initial symptom, and that one should be guarded in expressing a definite opinion too soon in the course of the disease, especially in children.

In every case of suspected gastritis, pregnancy, uremia, and the gastric crises of tabes must be excluded. Cyclic vomiting with acidosis in children should not be forgotten.

Treatment.—But little medical treatment is required in the majority of cases of acute gastritis. Rest in bed with abstinence from food until the nausea has disappeared is usually sufficient to effect a speedy cure. When there has been a gross dietetic error, and vomiting has not apparently been sufficient to empty the stomach, emesis may be induced either by draughts of salt water combined with the tickling of the throat, or, especially in hospital cases, hypodermics of apomor-

phine (grain $\frac{1}{8}$) may be employed. Free emesis usually shortens the attack and prevents intestinal complications. If the patient be accustomed to the stomach-tube, the stomach may be washed out by the ordinary method. A tube of fairly large caliber should be used to prevent blocking of the lumen by particles of undigested food.

If the patient is not seen during the early part of the attack, it is generally wise to administer a mild laxative, either calomel, or if the stomach is in a receptive mood, castor oil, magnesium citrate, or other saline aperient. If intestinal cramps and flatulency are present a high saline enema should be given. The nausea is usually relieved promptly after the taking of food has been discontinued. Nausea may also be relieved by small doses of cerium oxalate, or by minim doses of carbolic acid with or without diluted hydrocyanic acid. Such a prescription may be given as follows:

R—Acid. carbolic., 95 per cent.,
 Acid. hydrocyanic. dil. āā ℥xvj
 Mist. cretæ comp. ad ℥ij—M.
 Sig.—Teaspoonful in a little water every hour for four doses.

The following may also be employed:

R—Acid. carbolic. (Calvert's),
 Glycerin āā ℥ss
 Aq. laurocerasi ℥j
 Aq. menth. pip. ad ℥iv—M.
 Sig.—Teaspoonful in a little water every two hours.

The thirst may be controlled by cracked ice slowly melted in the mouth, or by $\frac{1}{2}$ -dram doses of creme de menthe in shaved ice, a remedy which is furthermore of great value in relieving nausea and vomiting.

It is rarely necessary, except in the food-poisoning cases, to continue abstinence from food after twenty-four to thirty-six hours. As soon as the condition of the stomach will permit, peptonized milk or milk with lime water, or cerium oxalate, may be given. In those to whom milk is distasteful, hot broths may be given. Solid food should not be given until nausea has ceased.

During the stage of atony that often follows an attack the bitter stomachics are of service. Of special value is the tincture of nuxvomica in 10-minim doses before meals, or the tincture of physostigma in similar doses. As, moreover, a condition of diminished gastric acidity usually follows an attack, diluted hydrochloric acid with the meals may be of service. This after-treatment, together with the limitation of liquids at the time of meals, and with a carefully selected bland diet, is to be continued until food is taken again with relish.

II. Infectious Gastritis (Food Poisoning; Ptomain Poisoning).—

Etiology.—Infectious gastritis may be induced through infected meat, fish or milk, or by vegetable poisons. Bacteria are largely responsible for the poisoning which follows the ingestion of certain food, because they have been identified in a large proportion of epidemics of food poisoning that have been scientifically investigated. The poisoning may be due to organisms that are present before death in the tissues of the animal from which the food was derived, or to those that have subsequently gained access to such food.

In most instances the infected food has been free from all objectionable odor and taste, and, contrary to the accepted belief, it is rather rare that the ordinary putrefactive organisms or their products are capable of causing poisoning in man.

The most important infecting organism is the *Bacillus enteritidis*, a bacillus intermediate between the *Bacillus typhosus* and the *Bacillus coli communis*, first described by Gärtner in 1858, and often associated with his name.

The epidemic investigated by Gärtner affected 58 of 93 persons who had eaten the meat of an ox that had been killed because it had diarrhea. Examination of the meat showed that its capillaries contained bacilli identical with those found in the tissues of the fatal cases.

The causative role played by bacteria in the production of gastro-intestinal catarrh is shown by an interesting experiment, made by Poels and Dhont.

A healthy ox was inoculated with a small quantity of pure culture of a variety of *Bacillus enteritidis* in the jugular vein; twenty-six minutes afterward it was killed and partially bled out. On bacterial examination a few colonies were obtained from the spleen and liver. None were found in cultures from the flesh; a few bacilli were present in the blood. After keeping a piece of the flesh at 20° C. for seventy-two hours the bacilli had become more abundant. The rest of the meat had been kept at a lower temperature, not over 5° C.; in this scanty bacilli were detected. "After we had assured the workers in the slaughter-house that we were convinced from the smallness of the number of bacilli present in the muscle that the danger of eating the meat could not be very great, and that it would probably cause only slight diarrhea, 53 persons resolved to eat it. Of these 53, 15 became ill in consequence; headache, gastro-intestinal catarrh, and abdominal pain came on in from twelve to eighteen hours; in some there was severe diarrhea."

The serum of typhoid fever agglutinates the Gärtner bacillus, showing a certain kinship between the two varieties.

Bacillus enteritidis in milk is not uncommon. Klein found this

organism in ten out of thirty-nine samples of milk. As boiling does not destroy the toxin, the ordinary methods of sterilization do not render the milk harmless.

Other bacilli, closely resembling *Bacillus enteritidis* morphologically, but differing from it in agglutinative properties, have been described by other observers.

A paratyphoid bacillus was found by Trautmann to be the cause of one outbreak, and in one epidemic the *Bacillus typhosus* was found to be the existing cause. In this instance the meat was taken from a cow with a splenic abscess that was found to contain pure culture of this organism.

In many instances infection by *Bacillus coli communis* has been the cause, less frequently the *Bacillus proteus*.

Special mention must be made of infection by the *Bacillus botulinus*, an anaërobic bacillus identified by von Ermengem in 1895, and found in diseased ham. This infection runs somewhat a different course from the Gärtner bacillus groups, and usually follows ingestion of diseased sausage meat. To this form of poisoning the name "botulism" is frequently applied. The *Bacillus botulinus* does not seem to develop to any extent in the human body, so that the symptoms, properly speaking, are not due to an infection, but to an intoxication by the toxin produced by the germ in meat.

In the case of *Bacillus botulinus*, the toxins are readily destroyed by boiling, but in the case of *Bacillus enteritidis* the soluble products are not thus affected, and hence meat containing such may be injurious even after it has been cooked.

According to Trautmann, food poisoning represents the acute, whereas paratyphoid fever infection—etiologically due to the same factor and having the same agglutinative reactions—represents the subacute type of infection.

Toxins of which tyrotoxin, found in various milk products, such as ice-cream, custard, and cheese, by Vaughan is an example, may be the cause for acute gastro-intestinal irritation. A variety of substances belonging to poisonous albumins have been isolated, but about these very little is accurately known.

Fish are infected by the same bacteria as meats. The roe of certain fish at spawning season is also poisonous to man. This is especially the case with Spanish mackerel.

Oysters are commonly infected by contaminated water in which they are placed to whiten them and to render them more palatable.

The ingestion of certain shell-fish, such as crabs and lobsters, are often followed in certain individuals by an attack of severe gastritis, which is, however, hardly to be considered toxic in character.

Symptoms.—The symptoms of food infection are those of a severe gastro-enteritis associated with those of a more or less intense toxemia. The intensity of the symptoms varies according to the quantity of the infecting agent that is taken, its virulence, and the susceptibility of the patient. The result may be anything from a slight passing illness, to a severe or a rapidly fatal attack. The onset may appear within a few minutes of the ingestion of the infected food, or may be delayed twenty-four to thirty-six hours.

Gastro-intestinal symptoms consist of nausea, vomiting, and abdominal pain. The vomiting is usually severe and frequent, and persists long after the stomach has once been emptied—quite a different picture from that observed in the ordinary forms of acute gastritis, in which vomiting usually ceases soon after the stomach has once been thoroughly emptied of its contents. Nausea and retching are severe and protracted. In rarer instances vomiting is absent, the symptoms being intestinal in character.

Diarrhea is usually present during the early stages, and is accompanied by severe griping pains. The temperature is usually somewhat elevated at the early stages and may be accompanied at its onset by headache, pain in the back and bones, and other manifestations of an acute infection. The duration of the febrile period is usually short, although in many instances fever may persist for days or there may be recrudescences from time to time. In these protracted cases the diagnosis from typhoid fever or paratyphoid may be exceedingly difficult.

Herpetic, urticarial, or erythematous eruptions are not uncommon. In rarer instances desquamation of the hands and feet may occur a number of days after the acute attack. Widely spread petechiæ have been observed.

Marked prostration accompanies even the milder cases, while severe infections are frequently ushered in by symptoms of collapse that may rapidly prove fatal. In these fulminant cases the collapse may appear before the gastro-intestinal symptoms have time to develop. In these cases the temperature is apt to be subnormal, the pupils dilated.

Pneumonic complications have been noted in a number of epidemics.

The urine is usually scanty and highly albuminous. In some instances there is complete suppression.

Special mention should be made of the infection symptoms of botulism. The onset is usually delayed until twenty-four to thirty-six hours after the meal, and is ushered in by nausea, vomiting, and severe abdominal pain. Diarrhea is not as commonly observed as in the cases due to infection by Gärtner's bacilli or the *Bacillus coli communis*.

Visual disturbances are observed in many instances, and consist of cloudiness of vision, dilatation of the pupil with loss of reaction to light, and ptosis. Burning thirst is usually present, but the swallowing of liquids is difficult and often leads to severe choking attacks.

Complete aphonia is not uncommon. The temperature is seldom elevated, and the pulse rarely rises above 90.

Convulsive movements of the muscles of the extremities is commonly observed; in severe cases there may be opisthotonos. Muscular paresis may occur, and dysphagia from paralysis of the esophagus has been observed.

Closely akin to botulism is the clinical course observed in certain cases of poisoning by methyl alcohol. A remarkable epidemic of the latter character has been recently described by Stadelmann,¹ in which the symptoms due to methylism seemed identical with those of botulism, except that no motor palsies were observed. Some of the victims died before characteristic symptoms could develop, for, unlike alcohol, the more deadly methyl spirits do not rapidly intoxicate, some twenty-four to thirty-six hours being necessary for the development of the syndrome. In this epidemic 130 cases came under Stadelmann's observation, of whom 58 died.

Treatment.—The treatment of food infections is that of a severe gastro-enteritis in addition to those remedies required to combat the depressive effect of the toxemia.

The stomach should at once be emptied, preferably by washing out its contents by the tube. In the event of the stomach-tube not being at hand, emesis may be produced by any of the ordinary means. Apomorphine, so commonly given in the cases of single gastritis, should be given with caution, if at all, because of its depressing effect.

Free bowel evacuations should be produced by salines, elaterium, or calomel. As these remedies take time for the desired effect, or may be vomited, colon irrigations or high enemas should be given at the earliest opportunity.

Collapse should be averted as far as possible by the judicious use of hypodermic stimulation. Camphor injections, caffeine-sodium-benzoate, and the preparations of digitalis are the most desirable forms of such stimulation. Strychnine should be given with caution in the cases that are complicated by convulsions or muscular contractions. Hypodermoclysis should be employed in the collapsed patients who suffer from repeated vomiting and severe diarrhea.

Albumen water, broths, and similar mild fluid may be given after nausea and vomiting have ceased, but not without caution even then, as relapses from premature feeding are not uncommon.

¹ Berlin. klin. Woch., January 29, 1912.

Convalescence is regularly protracted—the general strength is not apt to return for three to six weeks. The general management of these cases of protracted convalescence is practically that of typhoid fever.

MEMBRANOUS GASTRITIS (CROUPOUS GASTRITIS— DIPHTHERITIC GASTRITIS)

Etiology.—Membranous gastritis due to infection by the Klebs-Loeffler bacillus is of rare occurrence. Leary found but two instances of diphtheritic infection of the stomach in 136 cases of fatal diphtheria at the Boston City Hospital. Primary invasion is still more uncommon, as diphtherial gastritis is almost regularly secondary to a similar invasion of the esophagus, throat, or upper respiratory passages.

The lesion may be produced by infective agents other than the diphtheritic bacillus. It has occurred during the course of typhus fever, typhoid, puerperal fever, pyemia, and a variety of other infections.

Foulerton¹ reports a case secondary to quinsy, in which the infecting agents were the pneumococcus and the *Bacillus mesentericus vulgatus*. Dieulafoy reports similar cases due to the diplococcus. Other observers have found streptococci in the shreds of false membrane vomited by the patient.

Membranous gastritis may also occur when chemical irritants are taken accidentally or with suicidal intent. While no age is exempt, the disease is commonly observed in young children.

Pathology.—The changes produced in the stomach in this condition are not essentially different from those of diphtheritic inflammation of other mucous membranes.

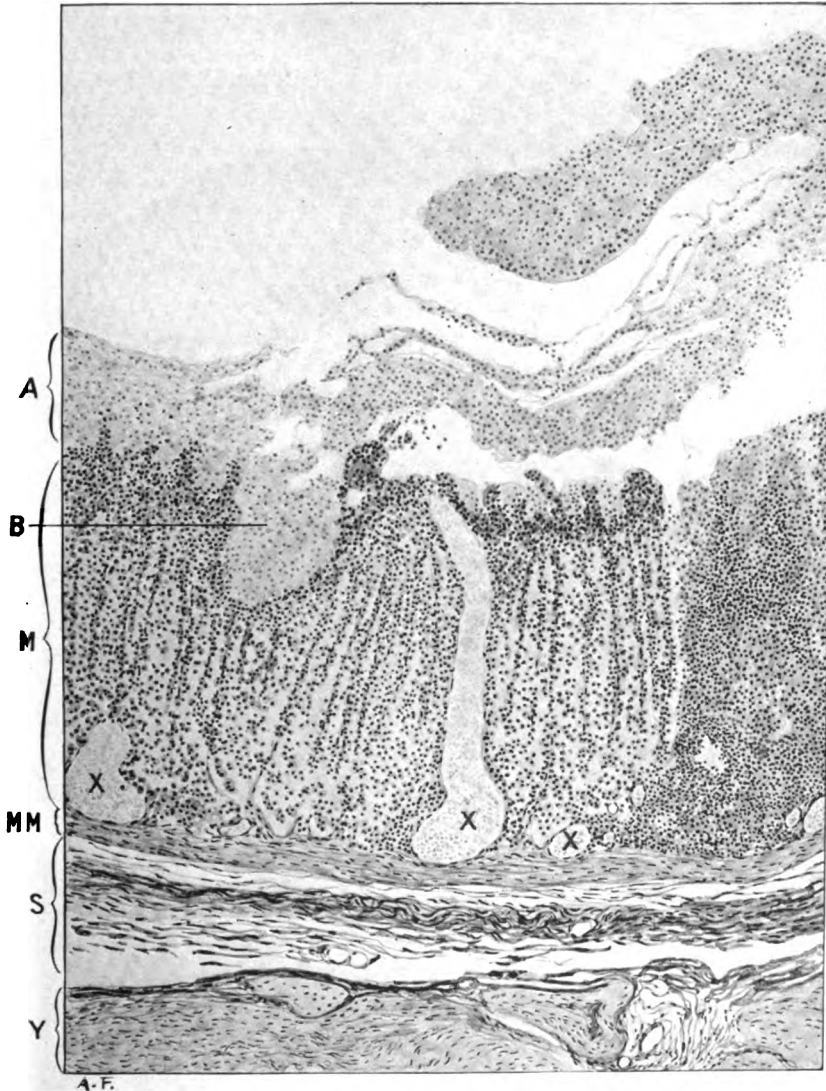
The mucosa is, at first, the seat of an acute inflammation which leads to degeneration and desquamation of the superficial epithelium. These degenerated and desquamated cells become mixed with fibrin, leukocytes, and red blood cells, all being fused together into a more or less homogeneous mass—the so-called false membrane—by the process of coagulative necrosis. In the mild cases this inflammatory exudate rests upon a fairly well-preserved mucosa and can be removed from it without much injury. When the process is more severe there is more or less extensive involvement of the mucosa, so that it is fused with the overlying membrane and the latter cannot be removed without injury to the former.

Generally the membrane occurs in irregular strips or patches. It may involve only the crests of the rugæ. On the other hand, cases

¹ Transactions of Pathological Society of London, 1902, p. 286.

have been reported where the membrane formed a more or less perfect cast of the interior of the stomach. The membrane is grayish in color or brownish from hemorrhage. The mucosa of the rest of the stomach

FIG. 1



Membranous gastritis. *A*, membrane, adherent to the mucosa at the left and dipping down into it at *B*. To the right the membrane has separated from the mucosa and appears coiled upon itself; *M*, mucosa showing dilated vessels *X, X, X*, and cellular infiltration to the right; *MM*, muscularis mucosae; *S*, submucosa; *Y*, muscularis.

is congested, and the gastric functions are even more markedly altered than in other forms of acute inflammation of the stomach.

There may be hemorrhage, generally slight in amount, from separation of the membrane. Necrosis and suppuration may occur, with extensive ulceration. However, as the disease is practically only seen in those already critically ill from some other affection, death usually ensues before such sequelæ have time to develop.

Symptoms.—Occurring usually as a late complication of severe infectious diseases, the superimposed infection of the stomach rarely furnishes any additional features to the original clinical course of the primary disease. There may be nausea and vomiting, but they are rarely sufficiently intense to attract attention to the gastric complication. There is nothing characteristic about the vomiting during the earlier stages of the malady. As the disease progresses, however, bits and shreds of the false membrane may be found in the vomited matters, and may furnish a clue to the underlying cause for the gastric complaint. The presence of false membrane in the vomited matters is not, however, pathognomonic of membranous gastritis, as membranous shreds may have their origin in the esophagus or throat, or be swallowed from a source higher up than the stomach, and then ejected from the stomach. Pus cells are usually found in the vomitus. During the stage of loosening of the membrane, hemorrhage may occur, usually insignificant in amount, although large, even fatal hemorrhages may occur.

Prognosis.—Prognosis is exceedingly grave. The gastric inflammation is not only serious in itself, but occurring as it usually does during the course of a primary infective disease of sufficient virulence to show a tendency to invade other parts of the body, the infection of the stomach is an additional pathological burden for the enfeebled system to carry. The pneumococcus infection seems generally less virulent than other forms of bacterial invasion. The prognosis of the diphtheritic form is more favorable when antitoxin treatment is given early. The disease is so rare and so seldom diagnosticated that the actual percentages of mortality cannot be given.

Treatment.—Treatment is directed toward the gastritis and toward the primary disease. The treatment of the gastric symptoms are those ordinarily adopted in every severe case of gastritis from whatever cause it might arise. The primary disease is to be treated in accordance with established usages. Bacterial vaccines may be given, according to the finding and identification of the infective organisms in the false membrane, but such vaccines unfortunately have to be given from stock preparations, as there is no time in each individual instance to work out the autogenous preparation.

PHLEGMONOUS GASTRITIS (ACUTE INTERSTITIAL GASTRITIS)

The term phlegmonous gastritis is used to characterize those cases of inflammation of the stomach in which the gastric submucosa, and to lesser extent the mucous and serous coats are uniformly or focally infiltrated with pus. The disease is quite uncommon, the number of reported cases being at the present time about one hundred. Men are more frequently affected than women, in the proportion of three to one.

Of 91 cases reported by Robertson in 1907, 61 were males, 19 were females, while in eleven the sex was not mentioned.

About one-half of the cases have occurred in day laborers—a class especially addicted to the abuse of alcohol and to excesses in eating, which tend to injure the lining membrane of the stomach. Alcohol may be regarded as a predisposing factor, although a history of alcoholism was only obtained in 14 out of 91 cases reported. Adams, in his reviewed cases, admits an alcoholic history in at least 25 per cent. of patients, although some have estimated that one-half of all cases occur in those addicted to drink.

The disease is not limited to any particular age, the youngest patient afflicted being ten years of age, the oldest eighty-five years. The majority of cases have occurred during the mid-period of life.

Etiology.—The primary cause is always microbic, the infection occurring either locally, through abrasions, or defects in the mucous membrane, or the invading germs may obtain entrance to the wall of the stomach through the blood current in various forms of septic disease.

Local infections of the stomach wall by pus-producing germs may occur through solutions of continuity of the mucous membrane, as in ulcer or cancer, or may follow operations upon the wall of the stomach itself. In 17 out of 91 cases the disease was preceded by chronic ulceration of the stomach. Solutions of continuity of the mucous membrane may also be produced by caustic agents, such as oil of turpentine or oxalic acid, or even by the use of drugs that cause intense inflammation of the mucosa. Klieneberger has reported one case that followed a dose of 45 grains of potassium iodide, in a patient who had not previously suffered from indigestion.

Abrasions of the mucous membrane caused by the contact of sharp, hard, scratchy substances introduced in the food may cause defects in the mucous membrane, admitting virulent bacteria. It may be impossible to find the point of entrance at the autopsy, but it is obvious that the pathology of infection lies through some abrasion of the mucous membrane, which cannot be demonstrated after death.

Infections through the blood current may occur in any pyemic condition. It was formerly a frequent complication of puerperal fever, and was especially marked in certain epidemics of which the epidemic of puerperal sepsis occurring in Prague in 1847 is perhaps the best example.

FIG. 2



Mucosa showing extensive infiltration of interglandular stroma with polymorphonuclear leukocytes. Destruction of lower ends of glands and the destruction of glandular elements in the more superficial portion by the inflammatory exudate.

Submucosa thickened, edematous, and infiltrated with leukocytes and fibrinous exudate.

In the 91 cases reported by Robertson, 11 followed the ingestion of improper food or poisons; 3 cases followed operations on the stomach; 2 occurred in pyemia, and 2 others followed trauma. In 1 case the disease followed purulent inflammation of the esophagus.

Pathology.—In the majority of cases the invading germ has been the streptococcus, often associated with the colon bacillus. Infection by the pneumococcus is not uncommon. In less frequent cases any one of the pyogenic organisms may be found.

Phlegmonous gastritis occurs in a diffuse and in a circumscribed form.

Diffuse Form.—The diffuse form occurs in 80 per cent. of all cases, and may affect the whole area of the stomach, or only a part, the pyloric portion being the most frequently affected. The wall of the stomach is greatly thickened, often attaining a depth of five or six times that of the normal. It occasionally attains a thickness even greater than this.

FIG. 3 ,



Phlegmonous gastritis. Section through stomach wall. The mucous membrane is seen much thickened at *M*, and with the muscularis mucosae on cross-section at *MM*. The greatly thickened submucosa which is edematous and infiltrated with pus is shown at *S*. *a*, muscularis. (From the Pathological Museum, Columbia University, New York.)

The essential lesion is a purulent infiltration of the submucous coat, which is greatly thickened and of a gelatinous or edematous appearance, or even containing channels or foci of liquid pus. These changes are nearly always more marked in the pyloric half of the stomach, and usually cease abruptly at the pyloric ring.

The muscular coat is affected to a greater or less extent and shows degeneration of the muscular elements. The mucosa may be normal in appearance. In other cases it may be acutely inflamed, and on its surface there may be hemorrhagic areas or necrotic ulceration. In a few of the cases the mucous membrane has been pitted by small apertures through which pus may be seen to exude.

Perigastritis almost regularly accompanies the disease, and a fibrino-purulent peritonitis occurs in over half the cases. Purulent pleurisy or pericarditis and abscess of the liver have been observed.

The duodenum is rarely affected. Involvement of the esophagus may occur, either secondary to the gastric infection or, as in the case reported by Pfister,¹ the esophagus may be the seat of the primary infection to which the gastric phlegmon is secondary.

¹ Deutsch. Archiv f. klin. Med., 1906, lxxxvii, 499.

Circumscribed Form.—The pathology of the circumscribed form is practically that of a localized abscess in the submucosa. The abscess may be single or multiple, and may vary in size from that of a hazelnut to that of the fist. Such an abscess is almost regularly accompanied by perigastritis, and is usually accompanied sooner or later by the lesions of diffuse peritonitis. Perforation may occur, either into the stomach, or peritoneal cavity or if limiting adhesions are formed, the pus may burrow into any near region. In one case rupture into the lung was recorded.

Symptoms.—Diffuse Form.—The symptoms of the acute diffuse form are those of a severe acute gastritis, running a rapid and fatal course, accompanied by signs of general sepsis, and frequently by general peritonitis.

The onset is usually abrupt, ushered in by a burning or gnawing pain in the pit of the stomach, and vomiting. A chill followed by a rise in temperature may be noted at the onset.

Vomiting is one of the most constant and distressing symptoms, appearing early and lasting throughout the disease. The vomited matters consist of ingested food, mucus, and bile. Pus that can be recognized as such by the naked eye is rarely found in the vomited matters, but pus to a microscopical degree is often present.

Boas and Adams both deny that pus is ever present in the vomit of the true phlegmonous gastritis, even when postmortem examination has revealed a sieve-like condition of the mucous membrane. The writer has, however, seen a case in which pus was evident to the naked eye.

Pain is an early symptom and is usually violent and continuous, although cases have been reported in which pain has been either slight or absent.

Tenderness may be present over the whole abdomen, but is more often confined to the epigastrium, although, as a rule, pressure over the epigastrium is not followed by an increase of the original pain, nor is the pain apt to be increased by moving or standing.

Fever is usually not high, ordinarily ranging between 99° and 102°. In rarer cases it may be as high as in other forms of sepsis, and may show a very considerable range of variation. The pulse is usually rapid and feeble, and is often out of all proportion to the degree of fever.

Prostration is a prominent symptom from the start, merging into collapse as a terminal event.

Mental phenomena are usually exhibited and consist of restlessness interrupted by periods of active delirium, which cease only when terminal coma intervenes. In rarer cases the mental faculties have been maintained until within a few moments of death.

Jaundice was present in 9 out of 41 cases reported by Leith.

When the peritoneum is involved the abdomen becomes tympanitic and diffusely tender. Prostration becomes more marked, and the pulse more rapid and feeble. The vomiting changes its character to that of a small intestine type, characteristic of peritoneal inflammation.

There are cases of diffuse phlegmonous gastritis that run a course exceptional in this, that the local symptoms are masked by those of the general infection. Fever of a pyemic type, with chills and occasional vomiting, may be the only symptoms present.

In the cases that complicate infectious disease of a septic character, the symptoms may be obscured by those of the original disease.

Circumscribed Form.—The symptoms of the circumscribed form are the same in type as those of the diffuse, although usually less severe and more protracted. Epigastric pain and vomiting appear suddenly, and are usually continuous throughout the disease, although they may become less marked as the disease progresses.

The fever assumes a hectic or pyemic type, and is usually higher than in the diffuse form. Drainage of the abscess into the cavity of the stomach is regularly followed by a reduction in the fever, as long as free drainage is established. In a few cases the fever may be slight or even absent, as in the case reported by Asverus.

Tenderness is present in the epigastrium, although it is less marked than one would expect. Rigidity of the muscles of the upper part of the abdominal wall is usually present.

Large circumscribed abscesses may rupture into the peritoneal cavity, causing speedy death, or rupture may occur through the mucosa into the stomach. In the latter case the vomiting of blood-stained pus may be followed by temporary improvement in all the symptoms.

In a case reported by Callow, twenty ounces of pus were vomited at one time, over and above the quantity that was passed in the stools. At the autopsy a quantity of pus estimated at seven pints was found in the peritoneal cavity. The disease had developed painlessly and without symptoms.

It is very difficult to determine when pus is vomited whether we are dealing with a localized phlegmon of the gastric wall or with a perigastric abscess that has ruptured into the stomach.

The clinical course of the circumscribed form may be protracted for weeks, with pain, vomiting, and signs of general sepsis.

Diagnosis.—Diagnosis is exceedingly difficult during life. No one has ever claimed to have made a correct diagnosis of the diffuse form.

In rare instances the circumscribed form has been recognized and successfully treated. The points on which the diagnosis is based are: An acute intense gastritis, local pain and tenderness, occasionally with

muscular rigidity over the epigastrium, or a palpable tumor, and a polynucleosis. Leukocytosis of 30,400 was found by Lengemann.

The differential diagnosis between localized gastric phlegmon and a localized peritoneal abscess following perforation of the stomach wall by ulcer or cancer is practically impossible.

Abscess of the liver may run a course almost identical in its symptom-complex. Acute pancreatitis should be considered a possibility in every case.

v. Leube reports a case in which all the symptoms of acute purulent gastritis were present, even pus in the vomited matters, but the autopsy showed a simple gastritis with a purulent secretion on the surface of the gastric mucosa.

Duration.—The course of the disease is rapid in the diffuse form, in which death often ensues at the end of twenty-four to thirty-six hours after the onset. More rarely the fatal issue may be delayed three or four days.

In the circumscribed form the duration is somewhat longer, often extending over ten days or two weeks, in rarer cases, longer than this.

Prognosis.—The prognosis is extremely grave, the mortality rate being about 98 per cent.

Deninger¹ reports the case of a female, aged thirty-two years, who complained of fever, violent epigastric pain, and tenderness, with diarrhea. A tumor as large as the fist was distinctly to be felt. On the nineteenth day she vomited a large quantity of pus, followed by a disappearance of the tumor, and recovery.

Glax² records the case of a young man, aged seventeen years, who suffered from severe pain in the stomach, and the vomiting of large quantities of pus, with gradual diminution of his symptoms. Recovery occurred in four weeks.

Mikulicz and Bovee³ have reported recovery after surgical exploration and the drainage of the cavity. All the cases of recovery to be noticed are those of the circumscribed form.

Treatment.—Aside from those very rare cases in which spontaneous recovery has apparently occurred, there is very little hope indeed, even by surgery, in effecting a cure in the diffuse form, and this difficulty is increased by the fact that in no reported case was the diagnosis clearly made before death.

In the circumscribed form, however, surgical intervention may be resorted to, and the abscess drained. Had the diagnosis of a localized

¹ *Deutsch. Arch. f. klin. Med.*, 1879, xxxvi, 624.

² *Berlin. klin. Woch.*, 1879, xvi, No. 38, p. 565.

³ *American Journal of the Medical Sciences*, 1908, cxxxv, 662.

abscess, in or about the stomach, been made, and if exploration and drainage had been resorted to, it is probable that the death rate would have been less appalling than it now is.

TOXIC GASTRITIS

The name toxic gastritis indicates a variety of lesions of the stomach produced by the taking of poisonous doses of chemical irritants.

Etiology.—The number of irritant poisons that may give rise to the condition is numerous. Among the most important are:

1. The concentrated acids, principally nitric, sulphuric, hydrochloric, hydrocyanic, carbolic, and oxalic acid.
2. Concentrated alkalies, such as lye and ammonia.
3. Irritant metallic salts, especially those of arsenic, copper, silver, and mercury.
4. The essential oils, such as turpentine, oil of Copaiba.
5. Certain metallic elements, such as iodine, bromine, and phosphorus, the latter being often an ingredient in rat poisons that are taken with suicidal intent.

Pathology.—The intensity of the damage done depends upon the character, the concentration, and quantity of the poison, the condition of fulness or emptiness of the stomach at the time the poison is taken, and the length of time the irritant remains in the stomach before it is ejected, washed out, or neutralized by the appropriate chemical antidote. Accordingly, we find lesions varying from simple hyperemia to ulceration, gangrene, or perforation.

In the mildest form there occurs a congestion of the mucous membrane, either diffuse or in localized areas, often with croupous exudations in patches, especially on the rugæ. There is usually a well-marked serous effusion into the submucous coat. Punctate hemorrhages into the substance of the mucosa may occur.

If the damage be more intense, areas of sloughing are seen surrounded by zones of intense congestion. In the case of even more concentrated and destructive poisons the mucous and even the outer coats may be converted into a pulpy mass of necrotic tissue, occasionally extending through the wall of the stomach so that perforation may ensue.

The appearance of the slough differs in different poisons. Sulphuric acid may produce grayish or grayish-white sloughs, later becoming dark brown as though charred. In less intense poisoning the parts may appear as if coated with white paint. Perforation is perhaps more common with sulphuric acid poisoning than with any other form of irritant, occurring in about one-third of the cases.

Pure carbolic acid is a good fixative for body tissues, and it may happen that should death occur soon after the ingestion of this poison, the stomach may look as if it had been boiled. The acid even may leak through the wall of the stomach and act caustically upon the peritoneum and even upon the spleen, and yet microscopical examination may show that the structures of the stomach are normal in appearance and well preserved. If the caustic action is less deep, the mucous membrane may preserve normal histological appearance, but underneath is a layer of dead or necrotic tissue.

Nitric acid usually produces sloughs of a yellowish color, resembling wet chamois leather. This acid is somewhat less corrosive than sulphuric acid. No case of perforation with poison by hydrochloric acid has been put on record.

Carbolic acid usually produces a whitening of the mucous membrane in patches, having a sodden appearance. Later the affected areas may appear dryish, as if tanned. The characteristic of hydrocyanic poisoning is the bright scarlet-red appearance of the slough. Alkalies are, as a rule, more destructive than are the acids, and the sloughs are softer, more gelatinous, and more widely spread.

The poisons act chiefly on those portions of the stomach and upper passages with which they first come in contact. The parts most affected are the lips and mouth, pharynx, the first portion and the lower portion of the esophagus, the pyloric end of the stomach, and pyloric antrum. The greater part of the esophagus usually escapes serious damage, although patches of ulceration, or even of gangrene, may be seen. While a general cauterization of the stomach may occur, the lesions are usually most marked in the pyloric portion.

As a rule, the entrance of the irritant into the stomach causes a sudden cessation of peristaltic action, so that cauterization does not usually extend below the stomach. In more rare cases, however, the poison obtains entrance in the duodenum or even the upper ileum, and produces lesions similar to those in the stomach, although somewhat less intense.

Should the patient survive the acute effects of the poisoning, the corrosive process is followed by the formation of granular tissue which results in cicatrization. If the affected areas be small, the defects may not be of serious import. When, however, the damage is extensive, the scar tissue may contract and lead to deformities such as hour-glass deformity, sacculations of the stomach, and pyloric stenosis. In other cases the mucous membrane of the stomach is replaced by thick fibrous tissue, which permeates the deeper coats, so that the stomach is shrunken and contracted, its lumen diminished, and its walls practically converted into dense scar tissue.

In the esophagus there are apt to be developed strictures, varying in extent and tightness, or the esophagus may become densely adherent to surrounding structures, especially the vertebral column, from which it cannot be stripped without tearing. Similar adhesions may bind the stomach to adjacent organs.

Symptoms.—The symptoms vary naturally with the intensity of the corrosive poison. In the most severe forms, such as follow attempts at suicide by concentrated acids, the taking of the poison produces a scalding pain in the mouth and the throat, and intense burning pain in the epigastrium. The agony may be well-nigh unendurable, and may induce a condition akin to surgical shock. The face is anxious, the pulse small and feeble, respiration rapid and shallow, the lips and face become cyanotic, and the patient may die of collapse with or without general convulsions. A fatal issue may occur within a few hours in extreme cases.

In instances of severe shock, such as accompany extensive corrosion, pain may be absent until a certain degree of reaction takes place. Vomiting may not occur until after the first symptoms of shock have passed.

Should the patient survive the initial symptoms of shock, the agonizing symptoms continue without abatement. Deglutition is excessively painful, often impossible by reason of pain and interference with the motor power of the esophagus. The soreness of the mouth and throat may be terrific, especially when the sloughs separate and leave bleeding ulcerations. Thirst is excessive and cannot be assuaged. Vomiting sets in after the acute stage of shock is passed, is incessant, extremely painful, and brings no relief to the distress. The vomiting is usually associated with continued retching. The vomited matters contain blood, mucus, and traces of the poison, thus facilitating the diagnosis in cases where the previous history is unobtainable. Shreds of mucous membrane and fragments of necrotic tissue may also be found.

The epigastrium is usually excessively tender to pressure. There may be fever, often obtaining the height of 104° . The urine is usually diminished, and contains albumin casts, and often blood cells, from an acute toxic nephritis induced by the poison. Total suppression may occur. After some days, usually between the seventh and the tenth, there may be vomited casts of the stomach or esophagus. Hemorrhage, often profuse, may occur at this time from erosion of one of the gastric arteries. Diarrhea with thin, bloody passages may occur, and symptoms of an acute ulcerative colitis may be added, especially after poisoning by corrosive sublimate.

In the case of certain drug poisons, such as phosphorus, symptoms of acute degeneration of the liver occur, continued nausea, intense

jaundice, and tendency toward hemorrhage. In such cases the symptoms of toxic nephritis are usually well-marked. Should extensive gangrene or deep destruction of the stomach wall occur the pain may become more generalized, tympanites and other signs of peritonitis supervene, and the patient dies within three or four days from perforation of the stomach.

If the effect of the poison be not great enough to cause immediate death of the patient, the symptoms may gradually amend in a few days, but improvement is slow and often incomplete. The patient may continue for days or weeks, with slight fever, pain, and vomiting, becoming more and more emaciated. From this condition he may gradually improve, or death may result from inanition and weakness.

Recovery may be incomplete because of extensive cicatrices that follow the healing of the ulcerated areas. There may be cicatricial stenosis of the esophagus, usually at the upper end, more rarely in the lower third or throughout its length. Pyloric stenosis may ensue. In some instances of excessive involvement of the stomach, the patient lingers for weeks with nausea, vomiting, and epigastric pain, and dies exhausted. In these cases the stomach is apt to be shrunken, and its walls thickened by the growth of dense fibrous tissue, which shows on its surface only here and there, traces of mucous membrane.

Diagnosis.—Diagnosis is readily made. The fact that poison has been taken may be elicited from the patient, or from friends, or some witness of the act. Search should be made for bottles containing the remains of the poisonous dose. Evidence of corrosive poison are usually visible on the lips, or in the mouth and pharynx, or upon the clothes of the patient. Further evidence may be afforded by the odor of the breath or the appearance and reaction of the vomited matters.

Prognosis.—The prognosis is always grave in the severer types of poisoning, and expression of opinion should in every case be given guardedly. Recovery is usually tedious and often incomplete. The danger of the resulting stenoses should always be borne in mind.

Treatment.—The first indication of the treatment is naturally to neutralize the poison that has been taken. This is preferable to early emesis if the proper antidote is at hand. Otherwise to avoid delay, the stomach should be rapidly emptied, by giving the patient large draughts of water or demulcent drinks, and by tickling the throat. This procedure is preferable to giving apomorphine.

For consideration of the proper antidotes the reader is referred to books on toxicology.

The use of the stomach tube as a means of emptying the stomach is said to be contraindicated, owing to the danger of perforation. That this danger does undoubtedly exist cannot be denied, but the risk run,

seems to the writer, to be less by the use of the tube than by allowing the poison to remain in the stomach even though proper antidotes have been administered, or by straining the stomach by repeated efforts at vomiting. Lavage naturally removes the poison more thoroughly than is possible by simple vomiting, and has the further advantage that the lavage water can be medicated by the addition of the chemical antidote. The removal of the poison by the tube, furthermore, minimizes the corrosive effect on the esophagus by a second transit through it of a corroding poison. For these reasons the writer believes it is much better to run the risk of perforation, and to wash the stomach gently but thoroughly.

After the stomach has been emptied, further attempts at vomiting should be controlled by opiates or anodyne, preferably administered hypodermically. Collapse and shock are to be combated on the usual principles. Hot fomentations over the epigastrium are of service in mitigating the epigastric pain, and demulcent drinks, with or without the addition of bismuth subnitrate and orthoform, may be given, unless contraindicated by attempts at vomiting. Feeding the patient by the stomach is to be interdicted until the distressing symptoms subside, and until then rectal alimentation should be resorted to. Should diarrhea prevent the latter form of feeding, and should inanition threaten, duodenal alimentation may be advised, by the use of Einhorn's apparatus.

CHAPTER II

CHRONIC GASTRITIS

BEFORE the general adoption of gastric analyses the diagnosis of "chronic gastritis" was made indiscriminately in cases of continued dyspepsia characterized by nausea and vomiting, that could not be ascribed to ulcer or cancer, so that the disease was regarded as one of common occurrence. During late years refinements of diagnosis have shown that the disease is relatively more rare than was supposed. Of the writer's private patients suffering from digestive disorders, less than 8 per cent. were sufferers from this ailment, although its frequency cannot be ascertained with any exactness, owing to the large number of cases in which the disease runs a latent course.

Primary gastritis is much less common than the secondary form. In 1306 autopsies at the Royal Victoria Hospital, gastritis was encountered in 108, of which only 16 were not definitely secondary to other causes.

Pathological reports are frequently of little value, as microscopic examinations are not usually made, so that antemortem congestion, contraction of the viscus causing apparent thickening of its wall, polypoid excrescences and mammillated appearances of its surface that show microscopically no true inflammatory changes, have been roughly designated as gastric catarrhs.

Postmortem changes occur with surprising rapidity so that the appearance of the mucosa is quite different from that during life. It is often extremely difficult at the autopsy to say whether or not a gastritis is present.

Etiology.—Primary and secondary gastritis are recognized. The causes for the primary form embrace all forms of irritation of the stomach that are continued over a long period. The dietetic errors that may so result are legion. Food that is improperly cooked, unwholesome, and irritating in quality or excessive in quantity, is a prolific cause. Many cases follow rapid eating and insufficient mastication, or eating at irregular hours. Excesses in tea, coffee, and in the chewing of tobacco may be mentioned as frequent causes, especially among the lower classes.

Among other etiological factors may be mentioned the taking of irritating drugs and the abuse of purgatives. The drinking of large

quantities of iced water is a common cause. It is generally considered that smoking to excess may produce the disease, but the writer can find no clinical evidence to support this statement, although it seems evident that abuse of tobacco may result in many forms of neurotic indigestion or even in atony.

Overindulgence in alcohol is by far the commonest cause for gastric catarrh, especially the use of the more concentrated forms of alcoholic beverages, and may lead to the definite clinical type commonly spoken of as alcoholic gastritis. Excessive alcoholic habits are, however, by no means followed with any certainty by gastritis, nor is the intensity of the inflammatory process, if it should occur, proportionate to the degree of the alcoholic abuse. Some stomachs seem immune to alcoholic irritation, while others are excessively susceptible, and react to quantities of alcohol that may seem to be within the limits of ordinary temperance. Definite lesions of gastritis were found in but one-half of 40 cases of alcoholism autopsied at the Royal Victoria Hospital. In the case of an alcoholic tramp who was repeatedly treated at Bellevue Hospital for delirium tremens, and who died from wet-brain and pneumonia, the stomach was filled with formaline solution a few minutes after death, and examined microscopically. It was found to be so free from all evidence of disease that it could be demonstrated to a class in normal histology as an absolutely normal stomach.

Acute gastritis does not usually merge into the chronic form, except when recurrences occur at short intervals from repeated dietetic errors, so that the inflammation has not the chance to subside between the attacks.

Although in many of the patients, one of the above definite causes can be ascribed, gastritis is often encountered in those who have led blameless lives, and whose diet and mode of life have been simple and wholesome, and we are at a loss to account for its occurrence. The relation of the milder forms of bacterial infection to these unexplained cases of gastric catarrh, has never been satisfactorily investigated, although the occurrence of ulcers experimentally produced in dogs by feeding them with cultures of the *Bacillus coli*, and similar lesions following the use of various toxins, suggest that a chronic catarrh may result from mild bacterial poisoning, insufficient to produce actual ulceration.

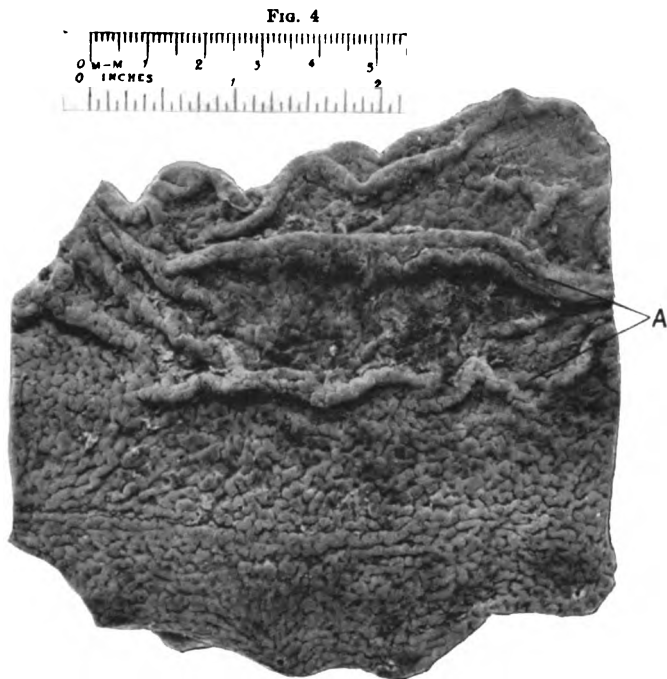
Secondary catarrh of the stomach occurs either as a complication of any gastric disease of a chronic character, such as ulcer, cancer, or pyloric stenosis, or it may be an associated lesion of chronic diseases of the heart, arteries, kidneys, or it may be concomitant with chronic pulmonary disorders.

The atrophic form may accompany cancer even if this invade a

distant organ, and often results from pernicious anemia. The writer believes that atrophy of the gastric tubules is rather the result of than the cause for this latter disease.

Senile changes of an atrophic character, especially marked in the prepyloric portion, are not uncommonly found in those aged over fifty years, and are one of the causes for indigestion in aged patients.

Pathology.—Chronic inflammation of the stomach produces a somewhat varied pathological picture and to the different appearances presented, various descriptive names have been applied. In general, two distinct types are seen; the one productive with an increase of connective tissue and more or less thickening of the stomach wall; the other, atrophic, with atrophy of the mucous membrane as the essential change. As in other inflammatory conditions of the stomach, the pyloric region is most apt to be involved, and in some cases the changes are confined to this location.



Chronic productive gastritis showing mammillated mucous membrane.

Chronic Productive Type.—The mucous membrane is indurated and gray, grayish red, or grayish violet in color. In many cases, especially those of long standing, there is a diffuse or patchy slate color from changes in the blood pigment. The mucous membrane tears less

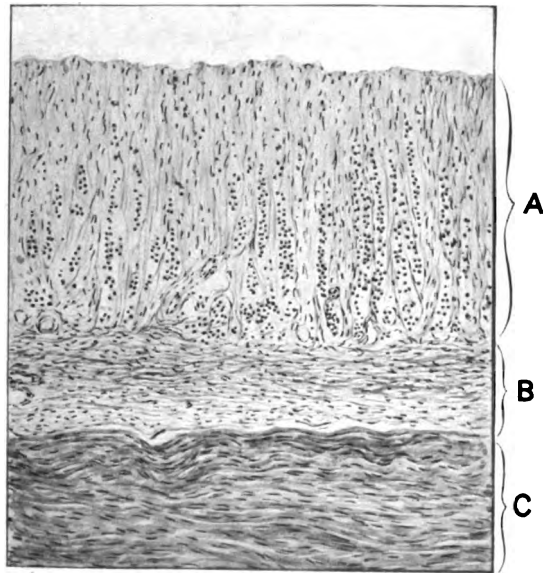
readily than normal, and it is not normally movable upon the underlying tissue. Occasionally small ulcers or superficial erosions are seen.

At times the thickened mucosa appears rough, wrinkled, and mamillated. It is to this condition that the French have given the name "état mammeoloné." Rarely this change is so aggravated that the term "gastritis polyposis" is fitting. These protuberances cannot be smoothed out by stretching as can the contractions of the normal stomach.

This type of gastritis may go on to a typical cirrhosis ventriculi, with very great thickening of the stomach wall, and a marked reduction in the size of the organ.

Microscopically, there is seen a proliferation of the interstitial and interglandular connective tissue, and of the glands themselves.

FIG. 5



Chronic atrophic gastritis. A, mucosa, showing marked increase in fibrous tissue with atrophy of the glands; B, submucosa; C, muscularis.

The thickened submucosa is firmly adherent to the muscularis which is generally very considerably thickened, the hypertrophy affecting principally the circular coat. This thickening may be so marked at the pylorus as to cause a certain amount of stenosis with subsequent dilatation of the stomach.

Even the serosa may be thickened and wrinkled.

The gland tubules in the mucosa may show irregular branching and

cyst formation. The latter may be so marked as to cause a true "gastritis cystica."

At times there may be seen in the midst of the mucous membrane, patches of epithelium and glands, identical with the intestinal type.

Atrophic Type.—The chief characteristic of this form of chronic gastritis, which is also known by the names "anadenia ventriculi" and "phthisis ventriculi," is atrophy of the mucous membrane with consequent destruction of the gastric glands. There is often an associated thinning of the other coats of the stomach with dilatation. However, as the condition is quite commonly secondary to the productive form, the mucosa may become extremely atrophic while the rest of the stomach wall remains very much thickened.

The mucosa is pale, opaque, and smooth, and the atrophy may be so extreme that scarcely a trace of glandular epithelium survives.

This atrophic gastritis is sometimes well seen in pernicious anemias as in the case described by Osler and Henry, in which the lining membrane of the stomach was "converted into a perfectly smooth, cuticular structure, showing no trace whatever of glandular elements.

Types.—Two forms of chronic gastritis are to be described:

1. *Catarrhal gastritis*, subdivided into:

(a) Those cases with hyperacidity.

(b) Those cases with normal acidity.

(c) Those cases with anacidity or "achylia."

2. *Alcoholic gastritis*.

CHRONIC CATARRHAL GASTRITIS

Clinical Types.—The division of chronic catarrhal gastritis into three classes depending upon the amount of gastric secretion present is not from any pathological reason, but simply because it facilitates the clinical description of the disease.

Of 350 cases of gastritis of non-alcoholic origin, observed by the writer in private practice, 40 per cent. showed hyperacidity, 24.5 per cent. showed normal or reduced acidity, 35.5 per cent. showed anacidity or achylia. In hospital practice the proportion of anacid cases was much higher.

These figures differ from those of McCaskey, who in 600 cases of gastritis found hyperacidity in 20 per cent., normal or subnormal acidity in 60 per cent., anacidity in 20 per cent. McCaskey, however, does not distinguish between the alcoholic and the non-alcoholic form of gastritis as does the writer. It will be shown later that in the alcoholic form, hyperacidity is not as frequent, and that normal or slightly

reduced acidity is much more frequent than in the non-alcoholic variety, so that after all the two sets of figures may not be as much dissimilar as they may at first appear.

I. Gastritis with Hyperacidity.—These cases are frequently alluded to as “acid catarrh,” or “hyperpeptic gastritis.” Clinically hyperacid gastritis presents itself in one of three different clinical types:

(a) *Acidity Type.*—The first group comprises those cases whose gastric symptoms consist of heart-burn and acidity. The patients are apt to be well nourished and of good appetite; they are not nauseated, neither do they vomit their food. Their chief complaint is that of heart-burn occurring at the height of digestion, one or two hours after food, for which they take soda, with instant relief. This feeling of acidity rarely if ever gives rise to a definite pain, a point of considerable importance in making a diagnosis between this condition and ulcer, the disease for which acid gastritis is most liable to be mistaken. In cases of ulcer without marked and characteristic pain, a differential diagnosis may be impossible, although the presence of occult blood in the stools may be of service in dispelling our doubts. *Persistent* intractable “hyperacid gastritis” usually turns out, however, to be chronic ulcer.

Acid catarrh may also resemble functional hyperacidity, especially as the symptoms are usually aggravated by overwrought and nervous states, and by stress of work, and are almost regularly less evident during freedom from business cares. Long continuance of the symptoms, absence of other neurotic manifestations, and the presence of mucus with evidences of imperfect digestion as shown by the gastric analysis point to an organic cause for the complaint. As an example of this type may be given the following case:

Male, aged forty years. For eight years has suffered from heart-burn, better in summer when he rests from work and takes daily exercise, than in winter when he leads a sedentary and harassing life. He is never without soda mints, which he takes almost regularly between meals. His appetite is good, he is well nourished and not anemic, and aside from his acidity has no other gastric symptoms whatever. He never has what he can call a pain.

Physical Examination. The stomach is of normal size and of good muscular power. Fasting stomach contains 25 to 30 c.c. of glairy mucus without vestige of food remains. Test breakfast shows a fine admixture of mucus of gastric origin. Total acidity, 86; free HCl, 60.

Lavage brings large quantities of thick, glairy mucus that can be lifted by a hook. By lavage and diet the symptoms disappeared and for a number of years he has remained well. Many cases of ulcer resemble this, but the writer believes the case to have been one of gastritis of the acid type.

(b) *Diarrhea Type*.—The *second group* includes those cases in which diarrhea is the prominent symptom and in which gastric distress of any kind is either insignificant or absent altogether. The evacuations are usually watery and profuse, and may occur with special frequency during the early morning hours. Mucus in the stools is rarely present, nor are there traces of occult blood, the absence of any evidences of catarrh pointing to an increase in intestinal peristalsis as a cause for the diarrhea. Carmine or charcoal, given by mouth, usually appears in the stools within ten to fifteen hours, indicating an abnormal rapidity in transit. Undigested particles of food, chiefly vegetable debris, may be found especially if mastication be not thorough. Undigested meat fibers are but rarely seen.

There may be abdominal distress or discomfort two to four hours after meals, often accompanied by borborygmi and distention, but actual pain either before or following the action of the bowels is quite uncommon.

The eating of hearty food usually aggravates the diarrhea, simple or semisolid food regularly reduces its severity. The ordinary treatment by astringents checks the disease but temporarily; sooner or later outbreaks occur. Considerable loss of flesh and strength result from the continued drain, and anemia often becomes quite pronounced. A typical case is as follows:

S. A., aged forty-three years, for three months has suffered from diarrhea, having from five to twelve loose stools a day, without blood or mucus. During this time he has lost twenty-seven pounds in weight, has grown so weak and anemic that he was referred to the writer as suffering from malignant disease of the colon. The diarrhea, while checked temporarily by drugs, soon returns as severely as before. The appetite is good, but as solid food regularly aggravates his trouble, he has been obliged to diet himself most rigidly, and has thereby added to the loss of his flesh and strength. He has neither pain, nausea, vomiting, nor any gastric distress, but he is annoyed by abdominal distention and uneasiness most of the time.

Examination shows a normally located colon of good muscular tone, and without any trace of catarrh. The stools are of thin pea-soup consistency, apparently well digested, and contain neither blood nor mucus.

The fasting stomach contains 25 c.c. of glairy mucus without free hydrochloric acid. Examination of the test breakfast shows a moderate amount of imperfectly chymified breadstuff mixed with thick ropy mucus of gastric origin. Total acidity, 85. Lavage brings large amounts of glairy mucus.

By lavage, diet, and small doses of bromides to diminish his intestinal

peristalsis, the diarrhea ceased in three days, his flesh and strength returned, and he has never had any return of his trouble.

These are the cases often treated for acute or chronic colitis. The rule should be, in every case of diarrhea, to examine the stools and to wash out the colon. If mucus in excess be not found, gastric analysis should at once be made, with a strong probability of finding the cause for the whole offending. It is interesting that the diarrhea in these cases cannot be differentiated from that due to achylia, except by gastric analysis.

(c) *Toxemia Type*.—In the third group are included those cases of acid catarrh—without gastric symptoms, but marked by severe recurring headaches and other manifestations of intestinal toxemia. The headaches are periodical, diffuse, although during the first few hours of onset they may be hemicranial in type, and are commonly spoken of as due to bilious attacks. Scotomas and fortification outlines do not commonly occur. Between these acute attacks there may be more or less constant dull feeling of oppression in the head, usually worse on waking and passing away by mid-day. Drowsiness and mental hebetude frequently are present to such a degree that mental or physical work is done only by the greatest effort of the will. In every case of “biliousness” or of recurring “bilious attacks” an examination of the gastric contents should be made.

Differentiation of Symptoms.—It is curious that in these three types the symptoms are sharply defined. Those who complain of heart-burn are not apt to have diarrhea or headaches, nor do those with diarrhea seem to be subject to heart-burn or intestinal toxemia. Those with headaches are not liable to suffer from gastric distress or diarrhea.

Aside from these principal clinical types, the writer has found that very indefinite symptoms were presented in about one-fourth of the cases in which acid catarrh is found by gastric analysis. There may be only a sense of oppression after meals, more marked after hearty and substantial food, or abdominal discomfort and distention, occurring at any time of day, but aggravated three or four hours after the taking of food. In a certain number of patients the only complaints were that they felt “run down,” and were constipated. There is nothing in the clinical history of such patients even to suggest acid gastritis. The diagnosis can only be made by the examination of gastric contents. The rule should be to examine the stomach contents of all patients with anemia and constipation, which prove rebellious to treatment. Many of these cases may turn out to be ulcer—only operation or autopsy can decide.

The hyperacid form is said by German writers to be especially common after abuse of alcohol, and to represent the initial stage of a

catarrh which, as the inflammation progresses, shows a reduction in acidity passing from hyperacidity to normal, from normal to subnormal, finally ending in total absence of peptic power or achylia. The writer ventures to express his doubts in this matter. Cases of hyperacid gastritis have been followed for years and never with any evidence of their ever showing such a reduction in their acidity, and, on the other hand, of many hundred cases of achylia, there was no evidence that hyperacidity had been the preceding condition. The writer believes that hyperacid cases remain hyperacid, and that subacidities represent a reduction from the normal without any preceding rise of acid values.

II. Gastritis with Normal Acidity.—Gastritis with normal acidity occurred in 24.5 per cent. of the patients with gastritis seen in the writer's private practice. It is probable that the actual proportion of normal acid cases is far greater than this, as digestive symptoms are usually so indefinite that the disease is unsuspected. There are no characteristic symptoms of this form of disorder. There is neither nausea, vomiting, heart-burn, pain, nor epigastric distress. Symptoms of intestinal toxemia appear but infrequently, usually occurring in those cases only that are complicated by a coëxisting atony, and are due rather to the atony than to the gastritis itself. This lack of symptoms of intestinal auto-intoxication is in striking contrast with the frequency in which toxemia occurs in the hyperacid and anacid cases. Diarrhea does not occur as in the hyperacid and anacid cases. Abdominal distention and discomfort are but rarely observed. One-fourth of the patients complained of a symptom which, however, is not distinctive—a peculiar gnawing, empty feeling in the fasting state. These patients become haggard and weary and worn unless they eat frequently. Similar subjective sensations, however, accompany the chronic forms of hypersecretion and are of common occurrence in the various forms of sensory neuroses of the stomach. In gastritis this sensation never amounts to a pain, nor is it usually referred to the epigastrium as a localized discomfort, but rather as a sense of general physical and nervous exhaustion.

Chronic gastritis with an acidity that is under the normal limits runs the same obscure clinical course as those in whom the degree of acidity is normal. Subacidity is, however, rarer than any of the other forms—downward gradation of gastric secretion does not seem to be gradual, and there are encountered comparatively few cases in which the acidity occupies an intermediate place between that of the normal and that of complete achylia.

III. Gastritis with Anacidity or Achylia.—The exact nature of achylia is quite obscure, although the disease is one of common occurrence,

being found by the writer in 1 out of every 16 private patients that apply for relief from dyspeptic symptoms. It may occur as a primary neurosis, or as the result of inhibition of gastric secretory reflexes, especially during the course of gall-bladder disease. It is frequently observed with cirrhosis of the liver, and often accompanies general arterial degeneration. It is one of the common causes for the dyspepsia of the aged, and results from atrophy of the gastric tubules in pernicious anemia and in cancer, whether situated in the stomach or elsewhere. It is obviously wrong, therefore, to describe all these various forms as the result of gastritis, for gastritis is but one of the causes that lead to a cessation of the function of secretion of the digestive fluid within the stomach. The presence of mucus in the test breakfast has been considered conclusive evidence of its origin in a gastric catarrh, but this is not a rule that can be carried too far. The dryish, squeezed-out, undigested breadstuffs that characterize the test breakfast in the majority of cases of achylia, when placed in water, show that mucus is generally disseminated in the undigested material, and suggests that the unchymified breadstuffs may act as a local irritant and provoke a flow of mucus, which, though scanty in amount, is designed to protect the mucous membrane from irritation. The writer includes under the term "chronic gastritis" only those cases of achylia in which the test breakfast shows undigested breadstuffs floating in an abundant sea of gastric mucus, as well as those instances of achylia in which lavage in the fasting state removes mucus in appreciable quantities.

Achylia thus considered occurred in 35.5 per cent. of cases of gastritis seen in the writer's private practice.

In hospital practice anacid gastritis is relatively more frequent than is observed in patients of the higher walks of life.

Symptoms of Anacid Gastritis.—The symptoms of achylia, the result of a chronic gastric catarrh, do not differ in any particular whatever from those seen in achylia resulting from any of the other causes. To avoid unnecessary repetition the reader is directed, therefore, to the symptoms of achylia in general, on p. 494.

Negative Symptoms of Gastritis.—**Appetite.**—Usually there are no changes in the appetite. The patients eat well and enjoy their food. If atony should exist as an associated condition there may be an early sense of satiety at the table, which renders it distasteful for the patient to eat sufficiently. In cases of gastritis occurring in neurasthenic subjects, the appetite may be capricious and fickle, and may even fail. In these two instances, however, the failure of appetite is due to the associated condition and not to the gastritis itself.

Nausea.—Nausea is not a symptom of gastritis. Neurasthenic subjects or those with atony may complain of a more or less constant

nausea, which is not influenced by eating, and which does not usually prevent their normal enjoyment of their meals after they are seated at table, but gastritis not thus complicated is but very rarely accompanied by nausea in the writer's experience. On the other hand, Elsner finds nausea in 40 per cent. of his cases.

Vomiting.—Neither is vomiting frequent in the non-alcoholic cases, although it is spoken of in the majority of text-books as frequent and quite characteristic. The patients are said to be nauseated after meals and to vomit small quantities of food intimately admixed with such tough mucus that the ejected matters stick tenaciously to the side of the bowl. This has not been noticed by the writer except after gross indiscretions in diet, sufficient to induce vomiting in anyone whose stomach is naturally sensitive, no matter whether or not chronic catarrh is present.

In those cases who overdrink or oversmoke, there may be morning vomiting of a brackish fluid that does not give hydrochloric acid reactions. This vomitus matutinus, as it is called, is simply the rejection of the stomach of saliva and pharyngeal mucus that has been swallowed during the night, and consequent upon chronic pharyngitis. It has nothing whatever to do with any morbid condition within the stomach itself.

Pain.—Pain does not ordinarily occur in gastritis. Exceptions to this rule must be made, however, in the rare cases of sclerosing gastritis with pyloric narrowing, and in a few cases of chronic atrophic gastritis in which lancinating pains of a neuralgic character may be a cause for complaint. Associated atonic conditions of the gastric wall may allow of gaseous accumulation, which gives rise to pain, but this is due to atony and not to the gastritis either directly or indirectly. Acid gastritis may be evinced by heart-burn, amounting often to distress, but this cannot properly be described as pain. *Eructions or accumulations of gas in the stomach do not occur in chronic gastritis that is not accompanied by atony, but regularly are present should this latter condition exist.*

Emaciation.—Emaciation is not common in gastritis, unless diarrhea be present or unless the diet has been insufficient. Progressive loss of flesh and an increasing anemia suggest the possibility of malignancy.

Excluding the rare cases of sclerosing gastritis in which gradual narrowing of the pyloric aperture occurs, chronic gastritis regularly runs its course without any diminution in the motor strength of its walls, or any retardation of the time in which the gastric contents leave the stomach. Vomiting of food too long retained does not occur, neither are present evidences of any of the forms of hypersecretion, either clinically or by examination of gastric contents.

Atony does not occur as the result of a gastric catarrh, no matter how intense the inflammation may be, but it is not infrequently observed as an associated and independent condition. When atony is present, gastric flatulence appears, giving rise to eructations and to gaseous accumulations in the stomach, often accompanied by some degree of distention pain. The appetite is easily satisfied, and the symptoms of intestinal toxemia become accentuated.

Diagnosis.—Gastric Analysis.—The diagnosis of chronic catarrh of the stomach cannot be made without the use of the stomach-tube. By gastric analysis many suspected cases prove to have normal gastric contents, and are to be classed among the neuroses, while in other cases, evidences of typical gastric catarrh are present when they are least expected.

1. Gastric analyses should always be made in every case of dyspepsia, no matter whether these symptoms be apparently gastric or intestinal, unless, of course, contra-indication to the passage of the tube exists.

2. Gastric analyses should be made in every case of chronic diarrhea that is not due to evident disease of the colon or rectum.

3. Gastric analyses should always be made in all cases of intestinal toxemia, of recurring headaches of toxic origin, and in patients who complain of the symptom-complex which is spoken of by the laity as biliousness.

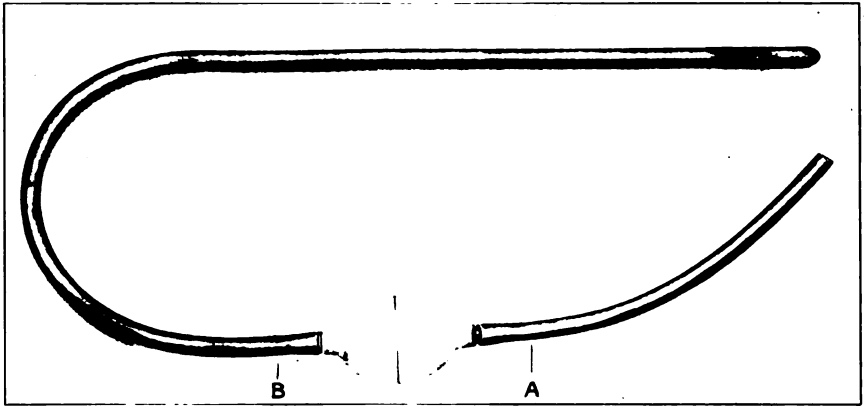
4. Gastric analyses should be made in all cases of anemia and general physical wretchedness without known cause and which are rebellious to treatment.

Examinations should be made both of the fasting and of the digesting stomach. The writer's procedure is as follows: The patient eats his evening meal as usual. Between 10 and 11 P.M. he is directed to consume a meat sandwich, preferably of roast beef, although any kind of meat will be satisfactory, and to drink a glass of water. Thereafter nothing is to be taken, not even a sip of water, until the following morning, when between 8.30 and 9 the tube is passed and the contents, if any, are withdrawn. No water is to be introduced at the time.

The patient is then given a breakfast roll, or an equivalent quantity of bread without butter, and a glass of water. One hour after the beginning of such a meal (not one hour after its completion) the tube is again passed and the contents of the stomach are drawn into an aspiration bulb (Fig. 6). The use of the bulb is preferable to the trusting of gagging efforts to force the gastric contents of the stomach through the tube into the receiving bulb. If mucus be present in the test breakfast the bulb is quickly detached and a larger one, shaped like a Politzer bag, that contains 200 c.c. of water, is substituted, the

water rapidly expressed through the tube into the stomach, and at once aspirated and the tube withdrawn. Withdrawal of the test breakfast should not take more than fifteen seconds from the time of

FIG. 6



Stomach-tube and aspirating bulb for extraction of gastric contents. Compression of the bulb with pinching the distal tube at *A* and then relaxing the pressure on the bulb serves as the vacuum. Releasing pressure on the distal tube, pinching the proximal tube at *B*, and compressing the bulb expel the aspirated contents into a graduate.

FIG. 7



Poltizer bag for experimental lavage.

the passage of the tube until its removal, and the experimental lavage by the method just described should not increase the length of ordeal more than an equal length of time. The reason why the writer recommends this experimental lavage is that in many cases of catarrh the contents of the stomach are so thickened by tenacious mucus that they are not readily withdrawn through a tube. It has repeatedly happened that the withdrawal of the test breakfast shows only a small quantity of apparently well-digested breadstuffs without mucus, while in the return of the lavage, mucus and food in large masses mixed with tenacious, glairy mucus are withdrawn. Should mucus appear with the experimental lavage given

in this manner, just after the withdrawal of the test breakfast, it is often desirable to ask the patient to return before breakfast on a subsequent morning for lavage—to determine whether mucus is secreted at all times, or only during the physiological congestion that accompanies digestion.

Gastric Analysis in Gastritis with Increased Acidity.—Examination in the fasting state may exceptionally show that the stomach is empty. Usually, however, there are withdrawn from 10 to 25 c.c. of a thin, viscid mucus, often slightly acid in reaction, although the presence of free hydrochloric acid cannot usually be demonstrated. *Hypersecretion is not a complication of chronic catarrh.* Food remains are not found in the fasting stomach even in microscopical proportions.

The muscular power of the stomach in uncomplicated gastritis is uniformly good, and signs of pyloric stenosis and food stagnation do not occur except with the stenosis form, to which special reference will be made.

Lavage usually but not invariably brings quantities of ropy mucus.

The test breakfast consists of breadstuffs more or less imperfectly chymified, and intimately intermixed with mucus. It is important to differentiate between pharyngeal mucus that has been swallowed and appears in the test breakfast and that form which is of gastric origin. Pharyngeal mucus is grossly and coarsely intermixed with food particles, is of a lumpy consistency, floats on the surface of the test breakfast, and can be drawn to one side in a thick mass by a hook. When it has been thus drawn aside the bulk of the test breakfast is disclosed of a more homogeneous consistency, and if gastric mucus be present, it will be seen to be uniformly and intimately admixed with the food particles. The floating pharyngeal mucus may be raised from the surface of the test breakfast in masses of considerable size, while gastric mucus cannot be elevated to any great distance by a hook. The test breakfast on standing does not usually separate into two layers, of clear fluid and of food debris, unless atony coexists, in which case the depth of the two layers is equal. More marked degrees of alimentary hypersecretion than this do not occur with gastritis.

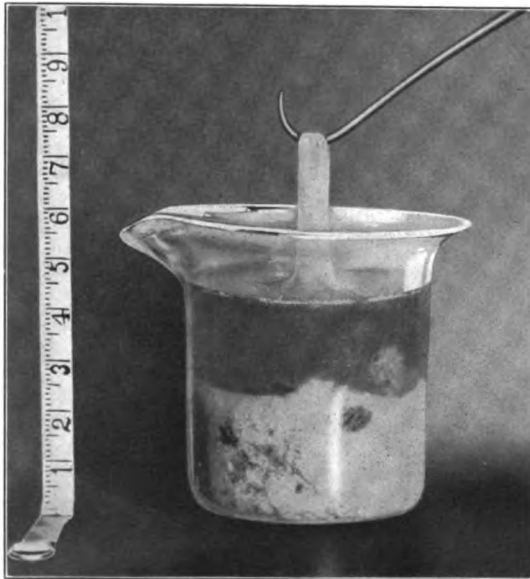
The total acidity ranges from normal to 75 to 85. Acidities higher than this are rare, and should suggest the possibility of ulcer. The proportion of free hydrochloric acid to the total acidity is practically that of the normal test breakfast. Occult bleeding does not occur.

The stomach contents of chronic gastritis are distinguished by the absence of organic acids—lactic acid does not occur. Many writers speak of various forms of organic fermentation encountered in gastritis, and of the presence of fatty acids and of lactic acid. This is entirely contrary to the writer's experience.

Lab-ferment is present. No conclusions can be drawn by the various methods of determining the strength of peptic digestion in these cases. Microscopically the presence of many leukocytes and epithelial cells of the gastric mucosa enmeshed in gastric mucus affords positive evidence of gastric catarrh.

Cohnheim and others describe the valuable aid in diagnosis afforded by the finding of bits of mucous membrane in the lavage water or in the test breakfast. The writer warns against placing too much confidence in the microscopic examination of these fragments, as too many sources for error are possible. These exfoliations are said to

FIG. 8



Test breakfast of chronic hyperacid gastritis. The supernatant fluid layer is of glassy mucus which can be raised on a hook. The bread particles are imperfectly chymified and flocculent.

be common in chronic gastric catarrh, although not confined to this disease. The writer believes that the greater the care taken to select a stomach-tube with openings that have smooth, rounded edges the fewer of these fragment bits are found. The sharp, punched-out apertures in many of the cheaper-made tubes often inflict traumatism on the gastric mucous membrane, which results in the cutting or tearing off of small bits of living tissue.

Gastric Analysis in Gastritis with Normal Acidity.—The examination of the fasting state and of the test breakfast is the same as that of the acid form, differing from it only in that the total acidity and free

hydrochloric acid are present in normal amounts. The lower the total acidity and the amount of free hydrochloric acid the greater is the reduction of peptic power, so that disks of albumin placed in the filtered gastric contents are dissolved slowly or not at all.

Gastric Analysis in Gastritis with Anacidity.—Achyilia.—In achyilia the fasting stomach is usually empty, although small quantities of gastric mucus may be found. The fact that motor errors do not exist is of the greatest value in differentiating this disease from cancer, in which evidences of stagnation either in the fasting or in the digesting stomach are found in nearly three-fourths of all the patients.

FIG. 9



Normal test breakfast. A moderate layer of supernatant fluid would have formed had the specimen been allowed to stand.

The test breakfast in achyilia occurs in any one of three forms:

1. The first and more frequent is the dry form in which the bread fragments have a dry, squeezed-out appearance, and look as if they had been chewed and spat out again.
2. The second variety is that of an apparently normal test breakfast, although it has not the smooth, purée consistency of the normal, but is more coarsely granular.

It is improbable that either of these two forms are found in achyilia that is due to gastric catarrh—they are seen in the other varieties of achyilia and are fully described in the article on that affection, in which the various types are discussed in detail (see p. 503).

3. The form of test breakfast that is indicative of gastric catarrh is known as the “wet variety of achyilia.” The test breakfast consists of poorly chymified breadstuffs floating in a sea of mucus, the quantity

varying from 2 to 8 ounces. The mucus may appear only in the contents of the digesting stomach, as is shown by the fact that lavage in the fasting state shows the stomach to be perfectly clean. The total acidity is negligible—hydrochloric acid in either free or combined form cannot be detected by chemical tests, and the peptic power of the filtrate is practically *nil*. Lab-enzyme is inactive. There have been attempts made to estimate the degree of damage done by the inflammatory process to the secretory apparatus of the stomach by determining the activities of the lab-zymogen. The following is a convenient method of testing quantitative reactions of lab-zymogen.

FIG. 10



Test breakfast in chronic anacid gastritis. The undigested breadstuffs are seen floating in the mucus, and settling leave a supernatant layer. To be contrasted with the normal test breakfast shown in Fig. 9.

One cubic centimeter of filtered gastric juice is introduced into a graduated measure of 10 c.c. capacity, water to the 10 c.c. mark is added, and the mixture is shaken several times. Five cubic centimeters of the admixture are then withdrawn by a pipette, placed in a beaker and marked "1 to 10." The examiner should now add water to the 5 c.c. which remain in the graduate until it again reaches the 10 c.c. mark. Five cubic centimeters of this mixture are to be withdrawn and marked "1 to 20." This dilution of the original 1 c.c. of gastric juice should be repeated and the dilutions marked "1 to 40," "1 to 80," "1 to 160," "1 to 320." To each beaker containing such diluted gastric juice are added 5 c.c. of milk and 2.5 c.c. of a 1 per cent. calcium chloride solution and the specimens set in a bacterial oven or water bath at 102° to 104°.

Normally, dilutions of 1 to 160 show a firm cake-like coagulation, dilutions of 1 to 320 are fine and flaky.

If in a given case of achylia normal coagulation by lab-zymogen occurs and firm coagulation appears with dilutions of 1 to 160, particularly if this be the result of repeated examinations, it is improbable that an organic affection of the stomach is present.

If the zymogen be diminished one-half in its activity a mild catarrhal process is probably present, and restitution by appropriate treatment is to be expected.

If zymogen reaction be absent in dilutions of 1 to 10 or 1 to 20, there probably exists a grave and usually incurable catarrh or atrophy of the gastric tubules.

Course and Duration.—The duration of chronic gastritis may extend over years, and although a “clinical” cure is to be expected, and the patient relieved of all distress, a *restitutio ad integram* cannot be hoped for. When the existing cause is found in some gross and palpable error in diet or in the habits of life that can be corrected, the greater are the chances of relief when these etiological factors can be eliminated. When there are no bad habits to throw away we are working more in the dark, and our results are not quite as good. The most favorable cases for treatment are those of the acid form because the actual digestive power of the stomach is good; there are inflammatory changes present, but functional activity of the stomach is unimpaired. In subacidity and achylia the digestion is mainly carried on in the intestine, and much depends upon whether or not the intestine is in a condition to do its work. Enteritis, which often accompanies achylia, regularly prolongs the duration of the disease and retards the clinical cure. Subacid or anacid cases with the preservation of normal lab-zymogen reactions may generally be regarded as favorable subjects for treatment.

Treatment.—Prophylactic Treatment.—This is the most important of all, and consists in the correction of all causes which have had any influence in producing or aggravating the gastric catarrh. Dietetic errors must be detected and corrected regularly and a reasonable uniformity of the meals insisted on. If the patient should insist that he eats only the simplest and most wholesome food, and that he is regular and temperate in his habits, it is often well to have him keep for a week a written list of everything he eats and drinks and the actual time of his meals, to which may be added an account of what he does during the day. The physician can often obtain more real information about his patient by such a record than in any other way. Slow eating and perfect mastication are to be insisted on, and all defective conditions of the teeth that render mastication difficult should be corrected. The teeth should be kept thoroughly cleansed, and

local applications made if Rigg's disease be present. Moderate smoking is not injurious, but it should not be carried to excess. The chewing of tobacco should be prohibited. If there be a misuse of drugs and carthartics, these should be either abandoned or greatly reduced, and the desired effect for which they were taken obtained in other less harmful ways.

Dietetic Treatment.—A number of diet lists have been recommended in gastritis, based upon the digestibility of the various articles of food included in the list, their bland non-irritating quality, and the short time they remain within the stomach before they pass through the pylorus in a perfectly digested state. Penzoldt, in conjunction with other clinicians, has elaborated a progressive diet for use in gastritis which complies with the above conditions, and becomes more liberal as convalescence is established. Penzoldt's progressive diet is as follows:

DIET I (ABOUT TEN DAYS)

Food or drink.	Largest amount at one time.	Preparation.	Special requirements.	How to be eaten.
Meat-broth.	250 gm. ($\frac{1}{4}$ liter).	From beef.	Without fat, not salted, or only a little.	Slowly.
Cows' milk.	250 gm. ($\frac{1}{4}$ liter).	Well boiled or sterilized.	Entire milk (or lime water $\frac{1}{4}$, milk $\frac{3}{4}$).	If desired, with a little tea.
Eggs.	One or two.	Very soft, just heated, or raw.	Fresh.	If taken raw it should be stirred into the warm, not boiling, meat-broth.
Meat solution (Leube-Rosenthal).	30 to 40 gm.		Should have only a slight meat-broth odor.	In teaspoonful doses or stirred into meat-broth.
Crackers (Albert biscuits).	Six.		Without sugar.	Not softened, but well chewed and insalivated.
Water.	$\frac{1}{4}$ liter.		Ordinary water, or natural carbonated water with a small percentage of CO (Selters).	Not too cold.

DIET II (ABOUT TEN DAYS)

Food or drink.	Largest amount at one time.	Preparation.	Special requirements.	How to be eaten.
Calves' brain.	100 gm.	Boiled.	Freed from all membranes.	Best taken in meat-broth.
Calves' thymus.	100 gm.	Boiled.	Likewise, especially carefully isolated.	Best taken in meat-broth.
Pigeon.	One.	Boiled.	Only young, without skin, tendons, and the like.	Best taken in meat-broth.
Chicken.	As large as a pigeon.	Boiled.	Only young, without skin, tendons, and the like (small fattened chicken).	Best taken in meat-broth.
Raw beef.	100 gm.	Chopped fine, or scraped, with only a little salt.	From the tenderloin.	To be eaten with crackers.
Raw beef-sausage.	100 gm.	Without addition.	A little smoked.	To be eaten with crackers.
Tapioca.	30 gm.	With milk, cooked to make gruel.		

DIET III (ABOUT EIGHT DAYS)

Food or drink.	Largest amount at one time.	Preparation.	Special requirements.	How to be eaten.
Pigeon.	One.	Broiled with fresh butter, not too much seasoning.	Only young, without skin, tendons, and the like.	Without gravy.
Chicken.	One.	Broiled with fresh butter, not too much seasoning.	Only young, without skin, tendons, and the like.	Without gravy.
Beefsteak.	100 gm.	With fresh butter, rare (English style)	The meat from the tenderloin, well beaten.	Without gravy.
Ham.	100 gm.	Raw, scraped fine.	Weakly smoked, without the bone.	With white bread.
Milk-bread or Zweibach or pretzels.	50 gm.	Baked crisp.	Stale (so-called rolls, etc.).	Carefully chewed, well insalivated.
Potatoes.	50 gm.	Mashed or boiled in salt water.	They should be mealy, and should crumble on crushing.	
Cauliflower.	50 gm.	As a vegetable, boiled in salt water.	Only flowers are to be used.	

DIET IV (ABOUT EIGHT TO FOURTEEN DAYS)

Food or drink.	Largest amount at one time.	Preparation.	Special requirements.	How to be eaten.
Venison.	100 gm.	Roasted.	Saddle, not too fresh, but without "hautgout."	
Partridge (quail).	One.	Broiled without bacon.	Young birds, without skin, tendons, legs, etc.; should hang for a time.	
Roast beef.	100 gm.	Medium to rare.	From good, fatted cattle; well beaten.	Warm or cold.
Tenderloin.	100 gm.	Medium to rare.	From good, fatted cattle; well beaten.	Warm or cold.
Veal.	100 gm.	Roasted.	From good, fatted cattle; well beaten.	Warm or cold.
Pike, shad, carp, trout.	100 gm.	Boiled in salt-water, without addition.	All fish-bones should be carefully removed.	In the fish gravy.
Caviar.	50 gm.	Raw.	Salted a little (Russian caviar).	
Rice.	50 gm.	Mashed, pushed through a sieve.	Soft, boiled rice.	
Asparagus.	50 gm.	Boiled.	Soft, without the hard part.	With a little melted butter.
Scrambled eggs.	Two.	With a little fresh butter and salt.		
Omelet (souffle).	Two.	With 20 gm. sugar.	Must have risen well.	To be eaten at once.
Stewed fruit.	50 gm.	From fresh boiled fruit, to be strained through a sieve.	Free from all kernels and peel.	
Red wine.	100 gm.	Light, pure Bordeaux.	Or some corresponding kind of red wine.	Slightly warm.

The disadvantage of such a diet is that although it is so simple and so easily digestible that it reduces the irritation of the inflamed mucous membrane to a minimum it is not sufficiently adapted to the needs of each individual case.

In every case of gastritis the motor and the secretory functions must be separately considered, and a diet should be individually and specifically ordered, which in quantity conserves the motor power, should motor error exist, and which in quality is capable of being digested by the digestive juices of that individual patient. Furthermore, the diet should be varied,

should be as mixed and general as possible, and should be practical for that patient to obtain and to continue in his own sphere of life.

Motor errors in gastritis rarely exist, so that food may be taken in fair and sufficient quantities. Frequent feedings need not be insisted upon in these cases.

When atony coexists, frequent small meals are indicated, the food should be given in as concentrated a form as possible, and liquids at meals are to be restricted. All motor errors contra-indicate the extensive general use of mineral waters as a means of cure.

More important is the regulation of the diet to accord with the *secretory errors* that may be present.

DIET IN HYPERACID GASTRITIS.—The diet in hyperacid gastritis should obviously be different from that advised in achylia. In the hyperacid forms of gastritis, the peptic power may be considered normal, and foodstuffs may be given in normal proportions, or even proteids and fats allowed somewhat in excess of the quantities that are usual in an average mixed diet. Meats that have a tough fiber are generally inadvisable, and should be replaced by those of more tender character, such as fish, chicken, fowl of any kind except goose, veal, lean ham, or tender lamb. All scratchy articles of diet are to be prohibited, and all overspiced and highly seasoned food. Smoking must be indulged in only after meals, and even then never to excess. Alcohol in any form is injurious, but if the patient be accustomed to its use, it may not be wise to cut it off altogether, but to allow a little whisky and water or a light, dry wine at the meals. Alcohol should never be taken between meals, nor at any time should champagne, sweet wines, or such heavy wines as Burgundy be allowed. Beer and ale are distinctly injurious.

Writer's Hyperacid Gastritis Diet.—Breakfast.—Coffee not advised; no tea allowed; no coarse cereal, such as oatmeal or cracked wheat; no bread crusts, dry toast, or hot bread; no salt fish or potatoes.

Allowed: Cocoa, with cream and sugar. Fine cereal, such as cream of wheat, farina, etc. Soft parts of bread, milk or cream toast. Crackers, thoroughly masticated. Butter, preferably unsalted, to be taken as freely as possible. Creamed or minced chicken; fresh fish; soft-boiled or poached eggs.

Luncheon.—Purée or cream soup of any kind, made without meat stock; no other soups allowed. Lamb; simply prepared ragout; lean broiled or boiled ham; fish, chicken, oysters in any form. Fowl, except domestic duck or goose. Mashed or baked potatoes; spaghetti or macaroni. Any vegetable that can be put through a purée sieve allowed. Any green vegetable (such as string beans) may be taken if tender, not if tough. Salad, with French dressing, made with

lemon. Farinaceous desserts, such as rice pudding, corn-starch, blanc-mange, custard, etc. No ice-cream or ices. No fruit of any kind. Alcohol not allowed in any form.

Cheese: Camembert, Roquefort, Cream, Brie, Neufchatel, pot-cheese.

Dinner.—Same variety as for lunch.

Between meals may be taken: Choice of custard, junket, raw eggs or egg-and-milk shake, chicken or meat sandwich; malted milk, cocoa. Milk in the glass not allowed.

Seasoning, such as pepper, salt, paprika, etc., should be reduced to the minimum.

Water should be cool but not iced; Celestins or Saratoga Vichy, Fachingen, Apollinaris, or Giesshuebler are preferable to plain water; when these waters cannot be obtained, may drink water containing one-fourth of a teaspoonful of bicarbonate of soda to the glass.

THE DIET IN GASTRITIS WITH NORMAL ACIDITY.—This differs from the preceding in one particular only—that proteid food should be given in somewhat smaller quantities. As a rule, meats should be allowed but once daily, as the peptic power in these cases is more or less diminished. An increase in fats is not to be advised.

DIET IN ACHYLIA.—The diet should consist theoretically and chiefly of carbohydrates and starches, with a greatly diminished amount of proteids and fats. Practically the diet can be made somewhat more liberal than this as intestinal and pancreatic digestion may compensate for the loss of peptic power. The full details of the achylia diet are given in a later chapter (see Achylia, p. 506).

Medicinal Treatment.—This may be administered either by drugs or by mineral waters.

The drugs that are of service in chronic catarrh of the stomach are very few, and their results are disappointing and uncertain. In the hyperacid forms, belladonna has been generally advised to reduce over-secretion. It should be continued in doses well under physiological limits for a considerable length of time. The writer has given this drug a fair trial, and has never been satisfied that it has done the least amount of good.

Silver nitrate may often be of service. It may be given in doses of $\frac{1}{2}$ grain, in distilled water before eating, or in pill or capsule. A serviceable prescription is as follows:

R—Argenti nitrat. gr. ss
 Ext. hyoscyami alc. gr. ss—M.
 Ft. caps. no. j.
 Sig.—One between meals.

Lavage with 1 to 3000 solution of the silver salt, every second day, may be given as in chronic indolent ulcer. *In very severe cases, especially if the diagnosis from ulcer is difficult, an ulcer cure should be advised.*

Alkaline Powders.—Alkaline powders may be used to neutralize the excessive acidity and to regulate the bowels. If the bowels are regular the basis of the powder should be sodium bicarbonate, to which sodium citrate may be added. If the patient be constipated, calcined magnesia should be added to the list of alkaline ingredients, while in cases of diarrhea calcium carbonate and bismuth subcarbonate are to be employed.

In hyperacid cases, olive or sweet almond oil or liquid paraffin may be taken before meals in teaspoonful doses. If preferred, oil can be given in the form of emulsion.

If hydrochloric acid be deficient, acids are indicated, either the dilute hydrochloric acid, or in the form of acidol tablets or oxyntin. Pepsin is often prescribed, as are the digestive ferments. Secretin has seemed to the writer to be of great service in these cases. The medicinal treatment of these cases of achylia is given in detail on p. 508.

Mineral Waters.—The treatment of gastritis by the use of mineral waters, either at home or at the appropriate "cure," is a form of treatment more in vogue in Germany than in the United States. It should be more frequently employed by us than it is, although there is no doubt but that better results are obtained at the "cure" than by the use of the same water at home. The freedom from business cares and worries, and the fresh-air exercise and relaxation, conduce greatly to beneficial results, but beside these evident advantages, mineral water fresh from the spring certainly has a more beneficial effect than the same water, bottled and imported, and vastly more than any form of artificial water made by the addition of powders compounded in the chemist's laboratory or obtained by the evaporation of the original water fresh from the spring. Nevertheless, bottled waters may be taken at home with advantage, and even may be artificially prepared. The choice of mineral waters should be carefully considered and never advised unless after due consideration of motor and secretory conditions. Motor errors, if present, contra-indicate their general use. Cases with atony should not be sent to drink the waters except in small quantities at a time, and only under medical advice; and in no case should more than a glass be given at a time, never more than three glasses daily, and never within two hours after a meal, nor one hour before the patient eats again.

The next important consideration is that of gastric *acidity*. There are three general classes of mineral waters that may be selected.

Carlsbad is indicated in gastritis with increased production of mucus, with high acidity. The water is to be given warm or hot, the hotter it is the less effect it seems to have upon the bowels. If constipation persists it may be given cool. Its prolonged use in cases which are persistently and obstinately constipated is not to be generally advised. The writer deprecates the giving of the water in doses sufficient to cause exhausting diarrhea, his rule being to give the water in such doses that the patient has but one unformed, but not watery movement daily. Smaller doses must be given at home than at the springs. Generally one glass as hot as can be sipped is to be taken before breakfast. If no looseness of the bowels result, an additional half dose is to be given at bedtime, or if necessary, one-half hour before lunch as well. If the one morning dose is too effective, only half the glass is to be taken in the morning and the remaining half at night.

The Carlsbad treatment should not be given longer than a month at a time without interruption. In those who are weak and debilitated Vichy may be given in similar doses, and of the temperature of 110° to 112°. Vichy and Carlsbad waters together with those of Marienbad, Franzensbad, and Tarasp are contra-indicated in all cases with diminished hydrochloric acidity.

Gastritis with diminished acidity is best treated by Kissingen (Rakoczy Spring), Homburg (Elizabeth Quelle), or Ems. The water is to be warm if the patient is constipated, hot if there be diarrhea. The dose should be one-half to one glass an hour before each meal. The results are exceedingly good when there is much mucus and hydrochloric acid is present, though in a diminished amount. Patients without traces of hydrochloric acid and without much mucus are not usually benefited by Kissingen water, or by the others just mentioned.

Patients with normal acidity are, as a rule, not benefited by medicinal waters, unless the quantity of mucus is considerable. The waters of Ems, Homburg, or Wiesbaden (Kochbrunnen) may be then recommended.

There are many medicinal springs in the United States which would rival in popularity those on the continent if the physiological results of their use on gastric secretion were as scientifically studied, and their respective indications in gastro-intestinal disorders as accurately defined.

Treatment with Lavage.—Lavage is a valuable adjunct of treatment in many cases, but it should not be indiscriminately advised. The writer's experience is that lavage is employed far too frequently in gastro-intestinal disorders and often does more harm than good.

The chief indication for lavage in gastritis is the *presence of mucus in such excess that it envelops the food masses and prevents their satura-*

tion by the digestive juices. The time has passed when we wash every stomach in whose contents small amounts of mucus are found. In the hyperacid form we must further remember that a mucous coating of the lining of the stomach is nature's protection against irritation by food and by gastric juice of heightened acidity. It has been even suggested that the relief from discomfort that often follows lavage with silver nitrate solutions is due rather to the effect produced by inducing free mucus discharge that protects the mucous membrane, rather than to its cleansing effect. When mucus is not present, there seems to be no reason for washing the stomach, especially as in gastritis food stagnation and fermentation do not occur. Lavage in impressionable subjects may occasionally prove beneficial as a purely suggestive and psychical form of treatment.

The beneficial effects of lavage are especially observed in gastritis with a normal or diminished acidity, accompanied by abundant mucous secretion. In achylia with the "dry form" of test breakfast the results of lavage are entirely negative.

Should atony coexist, lavage must be given with some caution. Only small quantities of water, never over half a pint, should be introduced at any one time, and every effort should be made to withdraw as much of the water as possible, so as to leave the stomach comparatively empty. The difference between the total amount of water introduced and the amount returned as wash-water should never exceed 10 to 12 ounces.

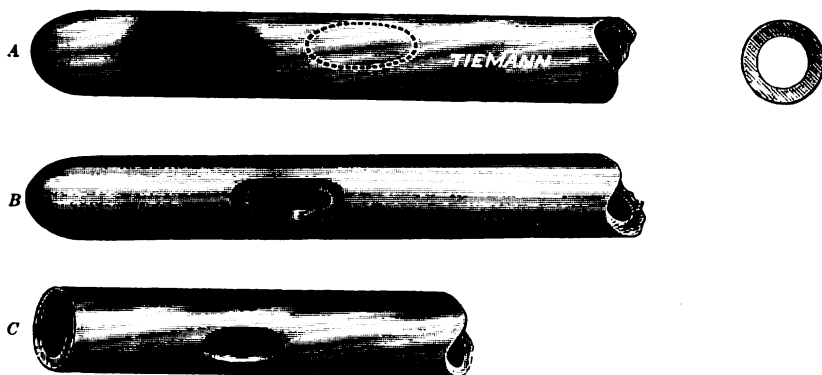
Lavage should not be given at a time when the stomach contains food, as the tube easily becomes blocked, and the amount of residual water retained is rendered excessive. It is much easier to pour water in than to siphon it out in these cases. Moreover, by lavage after meals the patient is deprived unnecessarily of his nourishment. The writer has repeatedly seen patients who wash their stomachs after each meal, because they found that mucus was present, depriving themselves of the benefit of their food, and inducing an atonic state of the stomach which aggravates their gastric distress. Lavage should never be employed oftener than once a day, and the writer's preference is in the morning before breakfast as a part of the morning toilet. In office practice the best time is late in the forenoon, at least three and one-half or four hours after breakfast. Lavage on retiring is often recommended, as it insures a clean and empty state of the stomach during the night; but when recommended at this time, it must be stipulated that the evening meal should be light, more of a supper than a dinner, and that at least three and one-half hours must elapse before the stomach is cleaned.

Care should be exercised in the selection of the tube. Tubes of small

caliber with small openings, and with rough edges to the apertures should be avoided. A fairly large tube of good caliber renders cleansing more effective, shortens the process, and is generally preferred by the patient.

The simple lavage set, comprising the tube, an extra piece of rubber tubing, and a funnel, serves admirably in the great majority of cases. When it is difficult, however, to withdraw the water introduced, the writer recommends that an aspirating bulb be inserted midway between the funnel and the glass connection that binds the extra rubber tubing to the stomach-tube itself. Pinching the tube on the proximal side with the fingers and compressing the bulb, and then pinching the distal side and relaxing the pressure on the bulb, will create a suction that is sufficient to aspirate the stomach contents.

FIG. 11



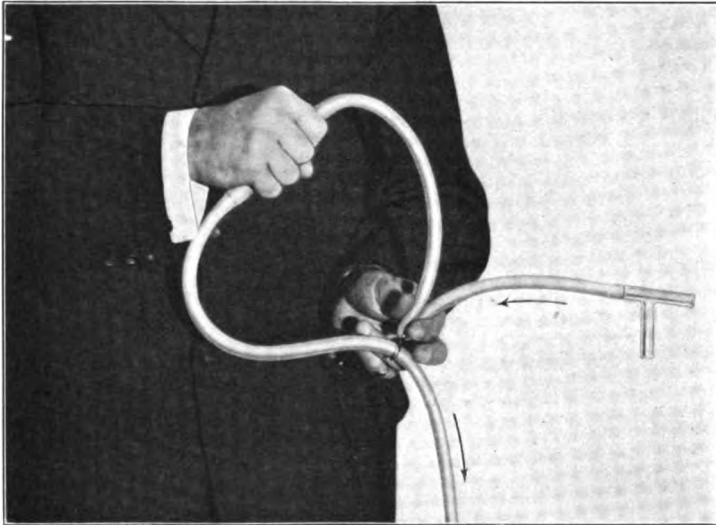
Stomach-tubes. *A*, proper tube, large smooth eye, sufficient caliber; *B*, improper tube, eye too small; *C*, improper tube, eye sharp and small.

The writer's apparatus for office lavage is thus constructed: A jar of 4 liters' capacity is connected by rubber tubing around with a pinch cork to a glass T-tube, to the remaining arms of which are attached the stomach-tube and a piece of rubber tubing that leads to a receptacle, graduated uniformly with the jar that contains the water to be introduced. An aspirating bulb is introduced in the exit tube near its connection with the glass T-tube, and the proximal and distal portions of the tube about six inches from the bulb are loosely looped together. The tube is held by the operator at the point where the tube is looped. Throwing the bulb to one or the other side kinks it so as to practically form a valve.

By alternately compressing and relaxing the bulb, and by creating kinks as described in one or the other loops, aspiration of the stomach

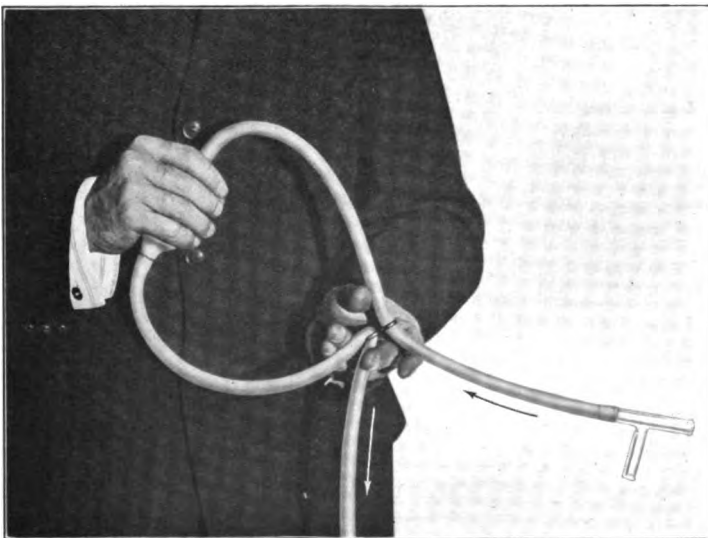
contents is rendered easy. By a reverse process water may again be thrown back into the stomach with sufficient force to dislodge adherent mucus, or to clean the tube should it become blocked.

FIG. 12



Expression bulb and valve. Flow from stomach blocked. Compression of bulb forcing its contents into the reception jar.

FIG. 13



Expression bulb and valve. Outflow blocked. Hand bulb aspirating fluid from the stomach.

ALCOHOLIC GASTRITIS

The alcoholic form of gastritis is characterized by the predominance of nausea and vomiting and by epigastric pain and tenderness. These symptoms occur but rarely in the non-alcoholic varieties. The impression that we have of gastritis, as a whole, largely depends whether we study the disease in private or in hospital practice, as in the hospital cases we have a large number of alcoholic patients, not only because the admissions are largely recruited from the laboring classes, in whom alcoholic excesses are unfortunately so common; but also because the symptoms of the alcoholic variety are more urgent than are those of the other forms, so that the patients are obliged by the necessity of their ailment to apply for hospital treatment. It is probable in this way that we have come to regard gastritis as regularly accompanied by pain, nausea, and vomiting. If we separate, however, those cases in which the alcoholic habit is not a factor in inducing the disease from those cases in whom it is well-marked and excessive, it will be seen that these symptoms occur almost exclusively in the latter class of patients.

Etiology.—It is implied by the term “alcoholic gastritis” that the cause is due to regular indulgence in alcohol, chiefly in a concentrated form. The cheaper varieties of whisky, largely consumed by the laboring classes, is decidedly more irritating than are the more refined brands, which they find too expensive to obtain. The difference in personal reaction to alcoholic irritation has previously been noted. Drinking on the empty stomach is more irritating than a similar quantity consumed at meals.

Pathology.—The lesions of chronic gastritis are almost regularly present with an increase in the amount of interstitial inflammation, more than is usually seen in the non-alcoholic forms. Owing to the predominance of the interstitial inflammation, there is a tendency for the lesion to assume the atrophic form. This was observed in 50 per cent. of the Bellevue cases.

In other and rarer instances the examination of the stomach fails to reveal a sufficient cause for distressing symptoms that have appeared during the life of the patient. The stomach may appear normal to the eye, while on microscopical examination the lesions of catarrhal or interstitial gastritis are so insignificant that it is difficult to explain the lack of proportion between the clinical symptoms on the one hand, and the pathological evidences of disease on the other.

Symptoms.—The symptoms usually appear after periods of excessive intemperance, and are relieved by the assumption of more temperate habits.

Nausea is an early and fairly constant symptom, appearing shortly after meals, and depending on its intensity upon the quantity of food that has been eaten. It is less marked when liquids or milk are taken than upon a general or mixed diet. With this nausea appears repugnance to food, which is often so marked that the patient refuses all nourishment, and attempts to sustain himself by repeated doses of whisky.

In more severe cases, vomiting appears after attempts at eating, and consists of food intimately admixed with ropy masses of mucus. Watery or acid-vomiting does not occur as in ulcer. The vomitus is not usually offensive, although evidences of fermentation may occasionally be present, especially if hydrochloric acidity be diminished. The vomiting does not cease when the stomach is empty, but attempts at vomiting continue, either as "dry retching" or resulting in the raising of small quantities of mucus or bile.

In the majority of cases there is now added the morning retching, which is one of the most characteristic symptoms of the disease; there are repeated gaggings and retchings which raise but small quantities of mucus or "slime," as the patients describe it, or else quantities of brackish fluid, which consists of the pharyngeal and salivary secretions that have been swallowed during the night. This latter form, the "vomitus matutinus," is rather an indication of chronic pharyngitis than of gastritis, but it occurs with unusual frequency in alcoholic patients. The morning nausea and retching are usually promptly relieved by drinking whisky, and the patient will often find that he is unable to eat his breakfast unless it is preceded by a dose of concentrated alcohol in one form or another. The taking of whisky before breakfast marks the height of alcoholic intemperance. Relief is often obtained by spraying the pharynx with a weak solution of cocaine (although this is never to be recommended to the patient, for obvious reasons) or by small doses of anesthesine placed upon the tongue and slowly swallowed.

Pain is almost exclusively seen in the alcoholic form, and is usually of a dull, aching character—rarely sharp or lancinating, quite different from the burning, boring pain of ulcer. From this latter condition it may furthermore be distinguished by its appearance during the *height* of digestion and not as in ulcer, during the emptying of the stomach; it is an "eating pain" and not a "hunger pain," occasionally quite intense, but more frequently described as more distressing than unbearable. In rarer cases the degree of pain is negligible. Tenderness is marked in the epigastrium during the acuteness of the attack. Tenderness on deep pressure is limited to the gastric boundaries, that elicited

by light pressure indicating tenderness of the abdominal wall itself, may extend beyond the confines of that organ.

Alcoholic gastritis appears usually in attacks, following alcoholic excesses and lessens rapidly with hospital care and a reduction of the exciting cause. During the stage of intermission the tenderness lessens and the nausea and vomiting disappear entirely, but are apt to recur from time to time, especially after dietetic errors. These symptoms slowly but steadily become less frequent and severe if the patient changes his habits, or may become again distressing after the next period of alcoholic excess.

Clinical History of Chronic Alcoholic Gastritis.—John T., aged forty-seven years; longshoreman; admitted March 28, 1909. For years the patient has been a steady drinker, taking whisky before breakfast, two or three drinks during the day, and as much beer as he can afford to buy. From time to time he goes on prolonged sprees, and frequently has been treated in the alcoholic wards for acute alcoholism. He eats irregularly, and his food is poor.

Three years ago he began to complain of morning nausea and retching, raising only a "little slime" after repeated gagging. He could not eat his breakfast until he had taken his whisky. This morning "dry retching" was especially marked after his sprees, and at these times there would be distress in the epigastrium, amounting often to a dull, aching pain, coming soon after eating, and gradually wearing away after one or two hours.

At the times of excessive drinking the pain was both severe and constant. He vomited but rarely except after his sprees, when he might go two or three days at a time unable to retain anything on his stomach. There had been considerable gas in the stomach and belching. Two weeks before admission he began to drink more than usual—his morning retching, nausea, and vomiting started anew, and he refused all food, taking only whisky. He became very nervous and unable to sleep, and entered the hospital complaining of nausea, vomiting, more or less constant epigastric pain, intense nervousness, and weakness.

Physical Examination. Well nourished; looks alcoholic rather than ill; arteries thickened, tension slightly increased; heart normal in size and action, second aortic accentuated; epigastrium diffusely tender; liver apparently normal in size; spleen not palpable.

Urine, 1030; trace of albumin, hyaline, granular, and a few epithelial casts.

Fasting stomach: Empty.

Test breakfast: 40 c.c., separating into layers; one-third clear fluid, two-thirds well-digested breadstuffs; no mucus; total acidity, 65; free hydrochloric acid, 30.

Diagnosis.—Gastric Analysis.—Examination of the fasting stomach is usually negative, although small amounts of swallowed pharyngeal and salivary secretions may be found. Hypersecretion and food stagnation do not occur.

1. The test breakfast may be unchanged to any great extent from the normal. It is remarkable what good-looking test breakfasts may be found in alcoholic gastritis with morning vomiting, and this apparent good digestion may even coexist with a clinical history of prolonged and severe outbreaks of the disorder.

2. In other cases the test breakfast is increased in amount, poorly chymified, and admixed with a large quantity of thin mucus. The acidity is reduced, although traces of free hydrochloric acid are present.

Estimation of pepsin acidity by the Hammerschlag or Metts method shows a reduction of peptic power. Proteid reactions are diminished. The starch digestion is usually carried to full completion. Occult blood does not occur. This tendency to the reduction of hydrochloric-acid secretion occurs more frequently in the alcoholic than in the non-alcoholic varieties. It is the writer's experience that a previous hyperacidity does not occur.

In long-continued cases we may find achylia, the test breakfast being either the "dry" or the "wet" form. In the dry form the test breakfast consists of a small amount of dryish, undigested bread fragments, as if bread had been partially masticated and then rejected. Mucus is present in small quantities, enveloping and infiltrating the food fragments. Hydrochloric acid is absent both in the free and combined states. Pepsin reactions are greatly reduced. This form of gastric analysis occurs but rarely in alcoholic gastritis. The following example may be given:

Mrs. X, aged thirty-two years, was well until 1897, when her husband suddenly died. She returned home to live with her mother, "who nagged at her," and she became so blue and depressed that she drank brandy continually "to cheer up."

In 1904 she married and was again happy, although she continued to drink "to be sociable." She soon began to complain of frequent attacks of vomiting, and her stomach pained her after her meals. These symptoms have continued with greater or less severity ever since—a period of nearly three years.

For the past four weeks she has awakened with nausea and "dry retching," and cannot eat until she drinks brandy, although she knows that if she drinks at this time she will suffer from nausea and vomiting throughout the day.

Physical examination showed no evidence of organic disease. Liver

and spleen apparently normal. Examination is difficult because of obesity.

The test breakfast brought 10 c.c. of dryish, squeezed-out bread-stuffs without admixture of mucus. Total acidity, 6; free hydrochloric acid, negative. The quantity was insufficient for estimations of ferment activity.

Wet achylia, or the form characterized by the overproduction of mucus, is more common than the preceding types. The test breakfast is more abundant, usually from 100 to 150 c.c. in quantity, and consists of undigested breadstuffs floating in this liquid mucus. Hydrochloric acid is absent. Peptic power is greatly diminished. Lactic acid does not occur.

Achylia in one or the other form regularly accompanies alcoholic gastritis when cirrhosis of the liver is present. In 14 consecutive cases of alcoholic gastritis, complicated by cirrhosis of the liver, this form of achylia was found in every instance. This is interesting, as it suggests that the achylia is the cause for the diarrhea so frequently observed in cirrhosis, instead of portal congestion as usually described, and that it is to be treated by entirely different methods and diets.

Prognosis.—The prognosis of alcoholic gastritis depends upon whether the patients are able to control their habits. If they will stop drinking, lead temperate and rational lives, a clinical cure will result, although it is not to be expected that the pathological changes in the stomach will show much if any improvement.

Treatment.—During the acute attack all alcohol should at once be discontinued. Should it so happen that the patient is on the verge of delirium tremens, bromides and chloral are to be given, preferably by rectum and ʒj doses of paraldehyde, two or even three times a day, are of service in allaying the restlessness. The unpleasant taste may be disguised as in the following prescription:

R—Paraldehyde	ʒj
Glycerin	ʒiij
Spirit vini rect.	ʒj
Tinct. cardamom. comp.	ʒj
Ol. aurantii,	
Ol. cinnamom.	āā m̄j
Elixir aurantii	ad ʒiv—M.

Sig.—Tablespoonful in water every three hours.

The occurrence of cerebral symptoms is not a sufficient reason for again resorting to the use of alcohol. Hot fermentations to the epigastrium are usually of the greatest comfort in relieving the soreness and distress. For a few hours the stomach should be given rest from food,

but abstinence should not be continued longer than ten to twelve hours. Liquid and semisolid food should then be given at intervals of two or three hours. The actual choice of food is quite unimportant; any simple semi-invalid form of nourishment will answer, as it is more desirable to see that nourishment is taken than to be overparticular. After the acuteness of the attack has subsided, more solid food can be given, and should preferably be rather highly seasoned.

Benefit often follows a stimulating carminative just before food is taken. Capsicum and nux vomica and digitalis are important adjuncts of treatment, and may be given as in the following prescription:

R—Tinct. capsici ℥iij
 Tinct. digitalis ℥v
 Tinct. nucis vomic. ℥x
 Tinct. cardamom. comp. ad 3j—M.
 Sig.—Such a dose in water before eating.

The following is often of service during the acute stages:

R—Tinct. capsici 3ij
 Sodii bromid. 3iij
 Elixir lupulini ad 3iv—M.
 Sig.—Dessertspoonful in water every two or three hours.

The bowels should be freely opened, preferably by magnesium sulphate. After the attack subsides the subsequent treatment is that of the non-alcoholic forms of gastric catarrh. Alcohol must be absolutely interdicted.

CHAPTER III

CIRRHOSIS OF THE STOMACH (LINITIS PLASTICA)

LINITIS plastica or cirrhosis of the stomach is a term used to designate a rare disease of the stomach characterized by a circumscribed or diffuse increase of connective tissue, chiefly in the submucosa, and to a lesser degree in the other coats, causing a marked thickening in its walls. A number of synonyms for this condition are found in literature; chronic interstitial gastritis, gastric fibrosis, sclerosis of the stomach, fibroid induration, hypertrophic stenosing gastritis, hypertrophic pyloric stenosis, submucosa sclerosis with chronic gastritis, and callous retroperitonitis are among those most frequently employed. The terms "linitis plastica," originally used by Brinton in his description of the disease, and "cirrhosis of the stomach" are the ones in most common use.

Nature and Pathogenesis.—There are many theories adduced to explain this peculiar condition of gastric fibrosis, few of them, however, are substantiated by facts. Among the various hypotheses advanced, only two, opposed to each other, are worthy of discussion: one that linitis plastica is a special lesion of indefinite nature and cause; the other that it is a variety of scirrhus cancer. The most important problem today is the distinction, if any, between linitis plastica and cancer.

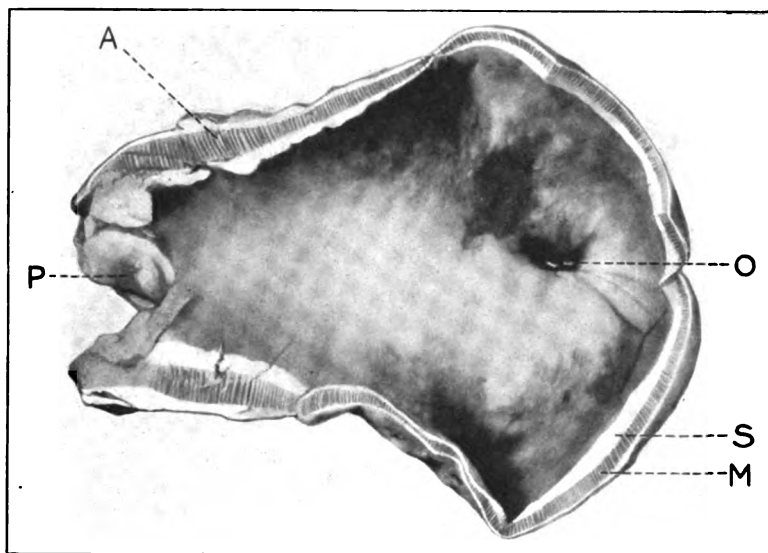
Before pathology attained its present stage of development, many cases were reported as benign fibrosis that would not bear muster under present requirements. Incomplete and hasty examinations in many cases have failed to reveal epithelial nests that probably would have been found had the search been sufficiently painstaking. Even microscopic examination, that seems sufficiently thorough, may fail at first to detect evidences of malignancy. More careful examination may finally detect cancer elements that were overlooked in the first examination.

Thus Curtis¹ exhibited the stomach, ileum, and colon of a patient as an example of linitis plastica. Discussion arising as to the completeness of this examination, he made a further search and found a small

¹ *Archiv. de méd. expér.*, 1908; also *Bull. et mém. de la Soc. anat.*, 1909, No. 1, p. 14; also *Bull. et mém. Soc. anat. de Paris*, 1909, No. 3.

area which was distinctly carcinomatous. Although the argument is not conclusive, it may be said that in a number of instances what seemed to be indubitable fibrosis of benign character at operation presented later clinical and pathological proof of malignancy. Woolsey, for example, reported a case of supposed linitis plastica, for which a gastro-enterostomy was done, with apparent restoration to health. Within two years, however, the patient died of cancer. It may be urged that in such cases carcinomatous degeneration occurred as a late event analogous to the development of malignancy at the base of a chronic gastric ulcer.

FIG. 14



Linitis plastica.

Those who assert that gastric cirrhosis is identical with a form of scirrhus cancer, support their view by the presence of analogous lesions in other parts of the alimentary tract, in the ileum, colon, rectum, peritoneum, and retroperitoneal tissues. These accessory lesions, considered by them to be due to a retrograde lymphatic involvement, suggest a cancer origin even though no cancer nests are actually discovered in the wall of the stomach itself. On the other hand, so-called cancer elements may be simulated by distorted glandular tubules infolded by dense masses of connecting tissue assuming somewhat an adenomatous structure, or by cells which result from proliferation of the endothelium lining the normal lymph spaces. It has been thought by some that the embryonic nature of the growth has been

suggestive of sarcoma. It is this uncertainty that makes it difficult to state without reservation that a true fibrosis of the stomach may occur without cancer. The existence of such a primary fibrosis cannot, at the present time, be denied, but it must be conceded that such a lesion is exceedingly rare, and that the great majority of cases reported as linitis plastica are really examples of cancer, the difference being that in the one instance cancer elements are found on microscopic examination, while in the other these elements are not found, either because they are overlooked or because they are not there.

Etiology.—The disease is one of adult life. Of 61 cases reported by Lyle the ages are grouped as follows:

Between 20 and 30 years	6 cases
Between 30 and 40 years	13 cases
Between 40 and 50 years	17 cases
Between 50 and 60 years	11 cases
Between 60 and 70 years	12 cases
Between 70 and 80 years	2 cases

Men are more frequently affected than women. In Lyle's cases there were 41 men and 22 women.

As to its actual cause we are entirely in the dark. Many writers claim that the disease is most common in tuberculous patients; other observers have attempted to refer the ailment to chronic venous congestion caused by arteriosclerosis and cardiac insufficiency. Both these etiological theories lack verification.

Alcoholism has been adduced as an etiological factor, but there seems to be no conclusive evidence that it stands in any causal relation to the disease. Syphilis may produce a connective-tissue proliferation in the wall of the stomach, which is with great difficulty distinguishable, microscopically, from the non-specific fibrosis. How many cases reported as examples of the latter affection may in reality have been specific is naturally a matter of pure conjecture. The localized form of hypertrophic pyloric stenosis of the adult (quite different in its pathology from that of the infant) may, according to some observers (Hemmeker, Boas, etc.), be not uncommonly caused by a chronic gastritis. It is exceedingly doubtful if this opinion be correct.

A few well-recorded cases have shown that the fibrosis had evidently started from the base of a chronic ulcer, the process practically resulting in a keloid.

It is an undisputed fact that ulcers, even though they be of small size, may result in the spreading formation of cicatricial tissue extending through the various coats of the stomach. Should the ulcer heal, it is naturally difficult to prove the etiological factor in such a sclerotic

process. When the ulcer remains open, the relationship between the ulcer and the spreading deposit of connective tissue is more obvious. Illustrations showing the relationship of ulcer to a localized fibrosis will be seen under the Section on Pathology. Multiple small ulcers have been supposed by some to be the cause for the widespread distribution of the lesion in some instances.

Pathology.—The disease occurs in a localized and in a diffused form.

Localized Fibrosis.—In this form there occurs a connective-tissue thickening from a localized induration in some part of the stomach wall. The favorite seat of selection is the pyloric portion, the new connective tissue often encircling and narrowing the canal. With but rare exceptions the infiltration ceases abruptly at the pylorus, and does not invade the duodenal tissues. In some cases the callous thickening is the direct result of an old ulcer situated in the pylorus. In other instances the ulcer may be situated at some distance from the pyloric canal, but from it, extending toward the pylorus, is an area of fibrosis, increasing in thickness as it approaches the orifice.

FIG. 15

Linitis Plastica.—No evidence of ulcer or carcinoma after serial sections were repeatedly examined. The block shows the actual size of the thickening. Note the abrupt change from the thick wall of the stomach to the thin wall of the duodenum. The muscular coat can be distinguished from the thin peritoneal coat and the greatly thickened submucosa. (From Bloodgood's collection of specimens at the Surgical Pathological Laboratory of the Johns Hopkins Hospital.)

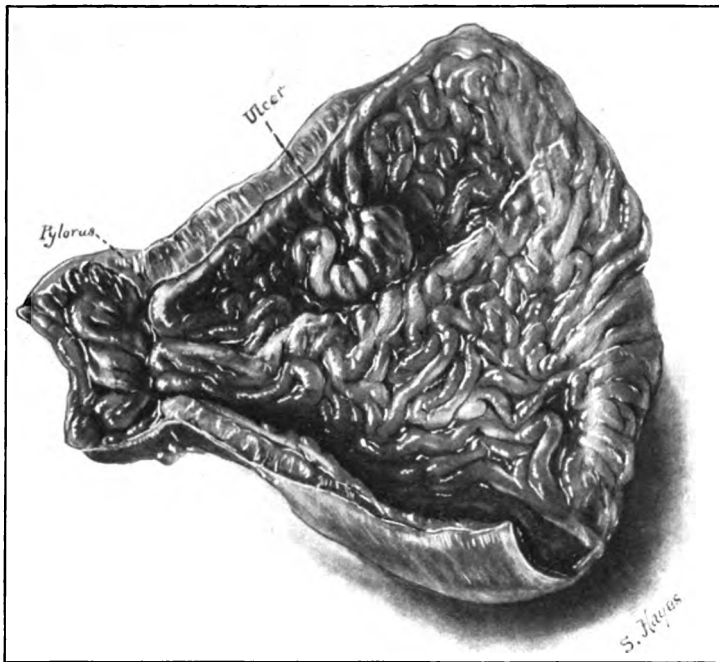


In other instances we have the same fibrosis of the pyloric end of the stomach, even to the point of extreme stenosis, without any evidence whatever of the previous existence of an ulcer. Whether an ulcer has previously existed or not is a matter of pure conjecture.

In the localized form with the lesion concentrated at the pylorus the tendency would naturally be for the stomach gradually to dilate. In some instances such an enlargement of the stomach actually does occur, so that the gross appearance of the stomach is that ordinarily observed in callous thickening of the pyloric canal from cicatrization of an old ulcer. It is probable that many recorded instances of supposed localized fibrosis in the neighborhood of the pylorus are in reality examples of this latter condition of cicatrized ulcer. In the majority of cases of localized fibrosis the stomach is unchanged in size. It may even be contracted.

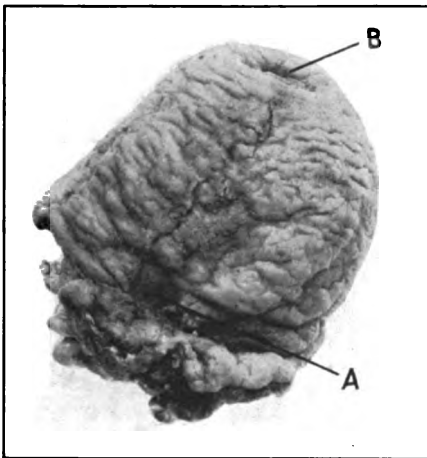
The localized form has been described by Boas under the terms chronic hypertrophic pyloric stenosis and chronic stenosing gastritis.

FIG. 16



Gastric ulcer on the lesser curvature some distance from the pylorus with circumscribed linitis extending from the ulcer to the pylorus. (From Bloodgood's collection of specimens in the Surgical Pathological Laboratory of the Johns Hopkins Hospital.)

FIG. 17



Chronic sclerosing gastritis due to ulcer at a distance from the pylorus, causing stenosis. Illustration shows the pyloric end of the stomach turned inside out, so that the mucous membrane appears on the outside. The ulcer is shown clearly at A. The narrowed pylorus is seen at B. (From Bloodgood's collection of specimens in the Surgical Pathological Laboratory of the Johns Hopkins Hospital.)

FIG. 18



Section through pylorus, showing connective-tissue thickening and muscular hypertrophy of the wall. (From Bloodgood's collection of specimens in the Surgical Pathological Laboratory of the Johns Hopkins Hospital.)

Diffuse Form.—In the generalized form the fibrosis induration is more diffusely distributed. The stomach is usually much contracted, often being likened in size to that of the normal colon. In Jacobi's case the lumen of the stomach was that of the small intestine. In Lyle's patient the stomach was spherical and of the size of a goose egg. In rarer exceptions the stomach may be normal or even increased in size. In the writer's case there must have been considerable dilatation because the volume of the vomited matters often exceeded a quart. Such instances are, however, quite rare.

The serous coat is thickened and has a dull white, opaque appearance, instead of being clear and shining.

The gastrohepatic and gastrocolic omenta usually show thickenings, contractions, and opacities, and the same appearance may be noted in the peritoneum of the ileum, colon, and along the course of the rectum. Dense areas of infiltration may be felt in the retroperitoneal tissues, the so-called retroperitoneal callous of Hanot. Lymph exudate may appear on the intestinal coils; there may be fibrous adhesions. Ascites is not uncommon. Cirrhosis of the liver may coexist. The stomach affords considerable resistance to the passage of the knife through its walls, and often gives rise to a creaking sound like cartilage when cut. When opened the organ does not collapse. The cavity of the stomach is often extraordinarily small, so that in the extreme degrees of contraction the organ may be able to contain only two or three ounces of fluid.

The lesion consists in the deposit of dense connective tissue in the walls of the stomach, the submucous coat being most extensively involved, the muscular and outer coats to a less degree. According to Brinton the submucosa is ten to twenty times its normal thickness, the serosa and subserosa seven to ten times, the muscular, five to eight times, and the mucosa, two or three times. The total thickness of the wall may be from one-half to one inch. Despite the infiltration the layers remain quite distinct from each other, and there is often a marked contrast between a fairly healthy looking mucous membrane and the enormous thickening of the remainder of the gastric wall. The thickening is rarely uniform. It is usually more marked at the pyloric end, and may be absent at the cardia. The mucous membrane may appear normal, or it may be mammillated. In other instances it is thinned and atrophic.

The pyloric canal is usually densely infiltrated, resulting in stenosis to a greater or a less degree. In some instances the orifice may be apparently patent, though from the stiffening of its walls the orifice can neither contract nor dilate, forming thus a rigid opening which practically has the same effect as a stenosis in affording resistance to

the passage of chyme from the stomach into the duodenum, and also allows of a duodenal regurgitation.

Microscopic Examination.—The mucous membrane may be normal, although usually evidences of catarrhal gastritis are present, with a well-marked, small-celled infiltration between the tubules. The presence of the infiltrating connective tissue may cause constrictions, cystic dilatations, and other abnormalities of the gastric tubules. In rarer instances there may be atrophy of the tubules and their replacement by connective tissue, so that the glandular elements completely disappear.¹

The submucosa and outer coats are permeated by dense connective tissue, uniformly or in bands, as has been above described. The muscular fibers may undergo atrophy, or in cases of long standing, hypertrophy of the muscularis may be observed.

Endarteritis of the nutrient vessels is commonly observed, although it is not an essential part of the lesion. The perigastric lymphatic glands are usually enlarged and show the lesions of chronic fibrosis. In other instances there may be an increase of cellular elements suggesting malignant involvement.

Through the submucous and muscular coats are occasionally found epithelioid nests. It is often impossible to say whether these are really epithelial in character and therefore carcinomatous, or due to proliferation of previously existing epithelial cells lining the lymph channels, which normally exist in these situations.

The pathology of the associated peritoneal and intestinal lesions and of the retroperitoneal callosities is that of the stomach wall. It is unknown whether these accessory lesions are concomitant with that of the fibrosis formation in the stomach, or whether they represent, as some believe, a retrograde lymphatic involvement suggestive of a certain degree of malignancy.

Symptoms.—Localized Form.—When the lesion is concentrated at the pylorus, the symptoms are practically those of pyloric stenosis. The onset is ordinarily insidious and without characteristic symptoms. There is usually distress in the epigastrium at the height of digestion, most frequently occurring one or two hours after eating, of a dull character, or else sharp and colicky. Tenderness in the epigastrium at this time may be elicited. There may be gaseous eructations, or the raising of acid fluid so characteristic of closure lesions of the pyloric canal.

As the disease progresses the symptoms of pyloric stenosis become

¹ See Nothnagel, *Deutsch. Arch. f. klin. Med.*, 1879, Bd. xxiv, 352; and Henry Osler, *Amer. Jour. Med. Sci.*, 1886, xc, 486.

more and more marked, the vomiting becomes a more serious feature, and interferes with the proper nutrition of the patient. The vomiting is usually provoked by the taking of more than a limited amount of food. The vomited matters consist either of food that has been recently taken, or they may be abundant, foul, and represent the food remains that have collected in the stomach as the accumulation of several meals. The latter type is characteristic of pyloric stenosis, the former that of a stomach intolerant of the quantity rather than the quality of food. The distress does not occur at any typical time relative to the hour of meals. Occult blood is not detected either in the gastric contents or in the stools.

As time goes on, the increasing inanition becomes more noticeable, and the subsequent course, if not interrupted by appropriate treatment, is one of progressive weakness.

GASTRIC ANALYSIS.—The *fasting stomach* is rarely empty. In the early stages small quantities of fluid (20 to 40 c.c.), giving reaction for free hydrochloric acid, are withdrawn. In the later stages the fasting stomach contains not only this acid fluid, but also food remains of previous meals in greater or less quantities.

Test breakfast usually shows at first the characteristics of an alimentary hypersecretion, the withdrawn contents separate into two layers, the upper layer of clear fluid being more than twice the depth of the underlying layer of well-digested breadstuffs. The acidity is usually normal or slightly excessive. Sarcinæ may be present. The above gastric analyses are practically those of any form of benign organic pyloric stenosis, and do not differentiate the disease under discussion from pyloric stenosis of a similar degree due to the cicatrization of a pyloric ulcer.

In other instances, while the same evidences of a motor disturbance of the pylorus are evident, as shown by the finding of fluid or food remains, but the acidity of the gastric contents is quite different. The amount of hydrochloric acid progressively decreases and lactic acid becomes present, together with smaller amounts of various volatile organic acids. The test breakfast is offensive, as are the vomited matters. Large rod-shaped bacilli resembling the Oppler-Boas form of lactic-acid organisms are present.

It is this type of gastric analysis that was present in Boas' cases,¹ and so closely resemble cancer of the pylorus that a differential diagnosis is quite impossible.

This clinical type is quite rare. No case that conforms exactly with Boas' description has come under the writer's observation, although

¹ Arch. f. Verdauungskrankheiten, 1898, iv, 47.

instances of malignant disease of the pylorus have been frequent enough and have given the same clinical history. It is possible that Boas' cases may have all been malignant.

Generalized Form.—In a few instances the disease has run a latent course for a long period of time, as the condition has been found quite extensively developed in cases of death from other causes, without there having been any complaint whatever of previous gastric distress. In Viti's case death resulted from arteriosclerosis, without gastric symptoms; the stomach was small and thick-walled, but without stenosis of the pylorus.

As a rule, however, gastric symptoms appear early in the disease and run a progressive course, terminating in death unless the condition be surgically relieved. Sudden onset of symptoms is somewhat less usual.

Pain or distress is one of the early symptoms, varying greatly in different cases both in intensity and in the time at which it appears in relation to the taking of food. Generally distress begins as soon after the meal, in proportion to the quantity rather than to the quality of what is eaten, and persists until the stomach partially empties itself either normally through the pylorus, or by the induction of vomiting. At first the patient can keep himself quite comfortable by reducing the quantity of food at each meal, but later the pain becomes more and more severe, and may appear after even very small quantities of nourishment.

Vomiting sets in with pain; at first only occasionally, but gradually it becomes more frequent, so that the patient will vomit part at least of everything he eats. The vomiting is characteristic of a small contracted, intolerant stomach that can hold only a certain quantity of food, rather than that due to pyloric stenosis with food retention.

In rarer instances the vomited matters are profuse, watery, and ill-smelling from the presence of organic acids, evidently the result of pyloric stenosis with food retention. This was the type of vomited matter in the case of the writer's, soon to be described.

Hematemesis and melena have been described, but are so rare that their occurrence should suggest malignancy rather than an inflammatory fibrosis of the stomach. There are at this time progressive anemia and emaciation, quite indistinguishable in character and degree from the similar cachexia observed in cancer. The vital powers finally diminish to the point of fatal asthenia. The final picture may be that of pernicious anemia, as in the case reported by Nothnagel and Henry Osler,¹ in which marked atrophy of the gastric tubules was found, together with the characteristic lesions of benign gastric sclerosis.

¹ American Journal of the Medical Sciences, 1886, xc, 498.

GASTRIC ANALYSES.—Gastric analyses are practically those of cancer. In the early stages of the disease the gastric analyses do not show any departure from the normal. When, however, the disorder is well advanced, there is a tendency for hydrochloric acid to diminish and finally disappear. With its disappearance there occurs the growth of Oppler-Boas bacilli and the formation of lactic acid. The examination of the fasting stomach may be normal, or may show a moderate degree of food stasis. Excessive amounts of fasting food remains are not ordinarily withdrawn at such an examination. Blood, either visible or occult, should not be present in either the fasting contents or in the test breakfast.

If it can be determined that the stomach will hold only a small amount of liquid, sometimes only a few ounces, the diagnosis will be supported.

Diagnosis.—Physical Examination.—During the earlier stages there are no characteristic physical signs. There may be a slight and diffuse tenderness in the epigastrium, more marked after the patient has eaten. It is only when sufficient thickening of the stomach wall occurs that resistance becomes appreciable, and finally assumes the definite character of a palpable tumor.

In the localized form the growth is over the site of the pylorus, and is quite indistinguishable from the mass found either with callous thickening of the pylorus from ulcer or with cancer. Adhesions, however, are less liable to occur than in these latter conditions.

In the generalized form the tumor becomes palpable, usually under the left costal arch, or lying obliquely downward and to the right in the epigastric area. It may be round or sausage-shaped, firm and smooth, fairly movable, and but slightly tender. Percussion over the tumor is never dull, but of a moderately resonant quality. It would, however, be the act of a bold, inexperienced observer to attempt to differentiate such a growth from cancer.

The size of the stomach is determined with extreme difficulty, owing to the resistance its walls afford to artificial dilatation. In any suspected case dilatation by the usual methods must be gradually and cautiously employed, as this method of examination may occasion exquisite pain. Usually all that can be said is that the stomach is not enlarged—how much smaller it is than normal is a matter of surmise, except by the *x*-rays, by which examination much valuable information can be obtained. In very rare cases the stomach can be made out enlarged in size, as in Nauwerk's patient, whose stomach filled two-thirds of the abdominal cavity.

Additional physical signs are afforded by the presence of ascites, by irregular fibrous thickenings along the course of the ileum, colon, and

rectum, and the retroperitoneal callosities, should these latter conditions be present.

The following case will exemplify not only the clinical features of the disease, but also the difficulties in determining the true nature of the lesion and its differentiation from ulcer or cancer.

Miss A. B. C., aged thirty-eight years, seen April 2, 1908. Except for occasional attacks of vomiting, extending over the period of one year, when she was a little girl, there had been no history of illness or of any gastric distress whatever, until six months ago, when she began to complain of the eructations of gas having a "rotten-egg" odor, and of occasional outbreaks of diarrhea. She began to lose flesh and strength, but has not had pain, nausea, or vomiting.

Examination.—Patient fairly well nourished and does not seem ill. A rounded, smooth, insensitive, and freely movable mass the size of a horse-chestnut is distinctly palpable one inch to right and one inch above the umbilicus. The lower border of the stomach by inflation is 4 cm. below the navel.

Gastric Analysis.—The fasting stomach contains 120 c.c. of well-digested food remains and fruit skins and seeds, separating on standing into two layers of equal depth. Total acidity, 50; free hydrochloric acid, 28; lactic acid, a trace; no occult blood. Microscopic examination revealed rod-shaped bacilli and some non-sprouting yeast, but no sarcinæ.

Test Breakfast.—220 c.c., well digested, separating on standing in two layers of equal depth. Total acidity, 64; free hydrochloric acid, 28; lactic acid, a doubtful trace. The diagnosis of pyloric cancer was made, and on April 22, 1908, Dr. W. J. Mayo excised three-fifths of the stomach.

Recovery from operation was uneventful. For twenty months the patient was free from all gastric symptoms, and except for some general lack of strength, seemed as well as ever. In February, 1910, after family illness with attendant worry and fatigue, the appetite became poor and she began to suffer from attacks of vomiting of acid fluid. These attacks came every three to five days, and were preceded for several hours by epigastric discomfort, which was completely relieved by the vomiting of about a quart of acid fluid and some food remains. Microscopically, the vomited matters contained a considerable amount of granular detritus, with numerous pus cells, some blood cells, and fatty acid crystals. Organized tissue could not be found. Bile pigment and lactic acid were present. Staining shows Oppler-Boas bacilli and non-pathogenic organisms, but pyogenic cocci could not be demonstrated on culture.

The fasting stomach contained 10 c.c. of greenish, viscid fluid con-

taining microscopical food remains and many Oppler-Boas bacilli. Total acidity, 10; free hydrochloric acid absent, lactic acid absent. No occult blood.

The test breakfast showed 10 c.c., yellow, pasty in appearance; traces only of gastric mucus. Total acidity, 10; free hydrochloric acid negative; no lactic acid; no blood.

The course from this time onward was one of progressive weakness. There was no actual pain, but continual distress increased by the taking of food. Every three to four days the discomfort would be more extreme, and would then be temporarily relieved by vomiting, as above described. Death occurred six months after the second accession of symptoms, a little over two and one-half years after the first onset of the disease.

There was considerable discussion about the exact nature of the lesion. The surgeon regarded it as malignant at the time of operation. The writer regarded the subsequent course quite characteristic of malignancy. Pathological reports varied. The pathological report at the time was "carcinoma (?)" with the additional note: "There is an adenomatous hypertrophy, but it is impossible to say whether or not it has begun to be carcinomatous." Another pathologist, equally eminent, reported: "The specimen is typical of cirrhosis ventriculi; the connective-tissue coat is much thickened; the mucous membrane intensely mammillated; the thickening of the connective-tissue coat at the pylorus is sufficient to give marked pyloric thickening. The glands are not involved, nor is there any evidence of peritoneal inflammation."

Differential Diagnosis.—*Cancer.*—The differentiation of cirrhosis of the stomach from cancer is well-nigh an impossibility. It is well, therefore, to regard the case as malignant until positive proof of its benign origin can be obtained.

Benign Stenosis.—In the localized form, in which the fibrous tissue is concentrated in the pylorus, with subsequent narrowing at that orifice, it may be difficult, or impossible, to determine whether we are dealing with such a condition, or with a callous thickening about the base of indurated ulcer in the pyloric portion of the stomach. Fortunately, however, such a differentiation is of very little importance, as the treatment of the two conditions by surgical means is the same.

Syphilis.—There are forms of diffused infiltration which histologically resemble very closely cirrhosis of the stomach, and which clinically run an almost identical course. In doubtful cases, therefore, a Wassermann test should be made, and remedies directed toward this specific affection should be given a fair trial.

Cirrhosis of Liver and Tuberculous Peritonitis.—When ascites is present linitis plastica may resemble cirrhosis of the liver, or tuberculous peritonitis. A careful study of the case in all of its aspects should ordinarily clear up the diagnosis without much difficulty.

Duration.—Duration is uncertain, as in many instances the disease becomes well established before it gives rise to symptoms. As a rule, the course of the disease extends over but a few years, two years being about the average period of time from the onset of symptoms until the fatal issue. Cases have been reported as having existed for ten years or more, but these are in all probability instances of callous ulcer of the pylorus, with dilatation of the stomach. In general terms the duration is that of cancer.

Prognosis.—Prognosis is most grave. Unrelieved by surgical intervention the outcome is invariably fatal.

Treatment.—Medical Treatment.—Medical treatment is entirely symptomatic, and is directed rather toward the reduction of distressing symptoms than with the hope of any possible effect upon the progression of the disease.

In the early stages some relief may be afforded by lavage, especially in localized fibrosis of the pyloric region. Here an indication for the washing of the stomach is to be found by the presence of food remains in the fasting state. There are patients who are apparently much benefited by lavage, even though the motor function is well preserved, and the lavage water returns clear. It is probable that the improvement in such instances is entirely psychic.

Artificial aids to digestion may be advised whenever hydrochloric acid is diminished, and the gastric digestion becomes thereby impaired. Dilute hydrochloric acid may be given with or after meals. As the capacity of the stomach is usually diminished, it is often better not to give the acid which requires bulk of dilution with water, but to prescribe oxyntin, with or without pepsin or acidol. The following may be recommended:

R̄—Capsule oxyntin with pepsin (Fairchild) gr. x

Sig.—Such a capsule with each meal.

The treatment of the vomiting is usually ineffective by medicinal means, as the vomiting occurs simply as the natural result of putting food into a indistensible contracted stomach of limited capacity. Small doses of strontium bromide may be given with or without codeine, but the greatest relief will follow the giving of a suitable diet.

Should the patient give a specific history, or should the Wassermann reaction be positive, a thorough antisyphilitic treatment should be administered. *Syphilis must always be considered a possibility.*

The principal rule for diet is to give highly nutritious food in small quantities at frequent intervals. The quantity suitable for each meal has to be determined by individual experience in each case. There is a point past which nourishment cannot be forced without resulting distress and vomiting, and the main indication for diet is to keep within these limitations. The quality of food makes very little difference as long as a nutritious selection is made.

Surgical Treatment.—As the disease cannot be differentiated from cancer, and as there is a growing inclination to regard all the cases as instances of malignant disease, the surgical treatment is that of actually proved malignancy.

Should the lesions be confined to the pylorus, partial gastrectomy should be attempted. The more thoroughly the diseased tissue is removed, the better.

Should the walls of the stomach be diffusely infiltrated, gastro-jejunostomy as a palliative operation is to be advised. The results of such an operation are often surprisingly good, a number of patients being reported in good health two or three and one-half years afterward.

If the disease has progressed so far that the cavity of the stomach is practically insufficient to hold enough nourishment to support the strength of the patient, or if the walls are too thickened and dense to allow of a proper juxtaposition with the duodenal loop, duodenostomy or jejunostomy may be the last resort.

CHAPTER IV

ACUTE AND CHRONIC ULCER

Gastric and Duodenal Ulcer.—In former years ulcerations in the upper alimentary tract were considered as being almost entirely gastric—duodenal ulcerations being treated with but scanty reference. In late years, however, duodenal ulcer has assumed a more prominent position in clinical pathology, and ulceration of gastric origin has shrunk somewhat into the background. It is largely to the surgeons that we owe our present state of knowledge on this subject, and we gladly acknowledge our great indebtedness to them for this and for other matters of gastric pathology.

It has become quite the fashion, however, to draw a too well-defined line between them, as shown by the large and increasing number of monographs entitled “duodenal ulcer” which appear in our periodicals, as if duodenal ulcer were an entity quite distinct from ulceration occurring on the proximal side of the pyloric ring. The writer concedes that gastric and duodenal ulcers may run their course distinguishable clinically from each other in the majority of instances—nevertheless there are cases of ulceration which it is clinically impossible accurately to locate at either the proximal or the distal side of the pylorus. We can say they are juxtapyloric, but farther than this we cannot go. Moreover, in pathogenesis, in pathology, in prognosis, and in treatment, it practically makes little difference where the ulcer lies. Embryologically the stomach and the upper four inches of the duodenum are derived from the foregut, and are therefore somewhat associated in function and pathology.

Accordingly, in the following article the ordinary forms of gastric and duodenal ulcers will be considered together. An attempt will, however, be made to differentiate them whenever they show differences in their pathological or in their clinical course, whenever such differentiation is of any real interest in the study of the subject.

An important distinction is to be made between the acute or “mucous” and the chronic or “indurated” ulcer. They differ from one another not only in certain details of their pathogenesis, but also in their location, number, size, and clinical history, and are radically different in their respective treatments. According to some authorities, the acute mucous ulcer does not apparently give rise to the chronic indurated form, but definite proof of this statement is lacking.

These differences will be noted throughout the following pages, whenever differences exist, without, however, separating the two forms from each other under entirely separate and distinct headings. It is believed that in this way their respective features will be more clearly contrasted and emphasized. Erosions and the rarer forms of gastric and of duodenal ulceration, as well as the jejunal ulcers which follow gastro-jejunostomy, will be considered under separate headings.

Frequency.—To determine the frequency of ulcer two methods may be adopted, both of which are open to serious objections.

The first method is to note the percentage of cases of ulcer which occurs in a large number of autopsies performed on those dying from a variety of different diseases.

The most frequently quoted figures are those of Welsh, which tend to show that gastric ulcer, open or cicatrized, occurs in 5 per cent. of all mankind. Fenwick found that either an open ulcer or a cicatrix was present in 4.2 per cent. of 47,912 autopsies. Small cicatrices are, however, easily overlooked, and, moreover, it is far more interesting to determine the frequency of ulcers that are unhealed at the time of death. It is evident that those who adopt the method of computing only those cases in which an actual ulcer is present will consider ulcer much less frequent than those who include in addition all the cases in which old cicatrices are present, and it is owing to this difference in statistical methods that the various estimates vary so greatly. Whereas, according to the first method, it may be said that about 4 or 5 per cent. of the entire population suffer at one time or another from the disease, it is probable that only 1 per cent. of persons dying from all causes show the presence of ulcer in a more or less active state.

Furthermore, ulcer occurs far more frequently in some countries than in others, being more common in Denmark and in northern Germany than in France or Russia.

In Copenhagen	ulcer was found post mortem in 20.00 per cent.
In Dresden	ulcer was found post mortem in 11.00 per cent.
In Tübingen	ulcer was found post mortem in 10.00 per cent.
In Zurich	ulcer was found post mortem in 2.20 per cent.
In Munich	ulcer was found post mortem in 1.23 per cent.

In the United States the frequency is much less than in Europe, as is shown in the following table:

Name of author.	Number of autopsies.	Number of ulcers.	Per cent.
Francine (Philadelphia)	2937	41	1.39
Kelly (German Hospital)	937	13	1.38
Howard (Hospitals of United States)	10,841	144	1.32
Mallary (Boston City Hospital)	2,600	25	0.90
Brooks (Bellevue Hospital)	1,000	9	0.90
Lockwood (Bellevue Hospital)	1,000	6	0.60

It is to be remembered, however, that these figures do not necessarily mean that in these cases the ulcer was the cause of death, but simply that at the time of death an ulcer was found. Neglect to appreciate this fact gives an erroneous idea of the mortality of ulcer. In 1000 autopsies at Bellevue Hospital, compiled by the writer, there were 6 cases in which ulcer was found. The cause of death in 1 case was perforation, in 2 cases lobar pneumonia, in 1 case bronchopneumonia, and in 2 cases nephritis, so that in only 1 of the 6 cases could ulcer be considered the actual cause of death. It does not seem, after all is considered, that these pathological statistics are of much value in determining the frequency in the *living* patient.

The second method of computing the frequency of gastric ulcer is based on clinical observation, and concerns the percentage of a large number of patients suffering from various diseases, who are diagnosed as suffering from ulcer. Such statistics must necessarily be very unreliable because of the impossibility of knowing whether or not the diagnosis has been correct. Many ulcers run an obscure or latent course, and are overlooked, while in others with gastric symptoms the diagnosis is incorrectly made.

Here, too, the personal equation of the observer must be taken into account. One clinician will be conservative and will include only the frank outspoken cases, while another will be less wary in making a positive diagnosis.

In general terms, however, it may be said that clinical experience leads us to believe that gastric ulcer is far less common than we might infer from pathological evidence. Lebert, in 41,688 patients in his care in Zurich and Breslau, diagnosticated the disease in only 252 cases (0.6 per cent.). Fenwick, out of 45,712 cases at the London and London Temperance Hospital in ten years, considered only 383, or about 0.8 per cent., as suffering from gastric ulceration.

Howard, in 161,599 medical admissions in various hospitals in the United States, found the clinical diagnosis of ulcer in 930, or 0.57 per cent.

In Bellevue Hospital from 1904 to 1908, out of 66,028 medical cases, there were admitted only 55 cases of gastric ulcer, and of these 11 were excluded by the writer as totally inconclusive.

The frequency of ulcer in private practice is often estimated by the percentage of ulcer cases that occur in those alone which are suffering from indigestion and gastro-intestinal disorders, and therefore the apparent frequency is somewhat greater. In the writer's private practice, 3 per cent. of patients complaining of gastro-intestinal disorders have suffered from gastric or duodenal ulceration. This is very close to Sawyer's report of 63 ulcers in 1800 gastric cases (3.5 per cent.).

On the other hand, Friedenwald makes a somewhat higher estimate, for in 12,598 patients complaining of digestive disorders, ulcer was diagnosed in 7 per cent.

Relative Frequency of Gastric and Duodenal Ulcer.—Ulceration of the stomach or duodenum may exist whenever unneutralized gastric juice comes in contact with the mucosa. Formerly gastric and duodenal ulcers were not as clearly distinguished from each other as they now are, partly from lack of care in locating them, and partly because, in the presence of ulceration or of extensive adhesions, it may be quite difficult to locate the pylorus accurately. W. J. Mayo has, however, called our attention to the value of the pyloric vein as a landmark. This vein extends from the inferior margin of the pylorus on its gastric side, upward, and across for three-fourths of an inch. A similar vein from above extends downward until it nearly or quite meets the one from below. Ulcers formerly regarded as pyloric are now generally considered to be of duodenal origin.

The relative frequency with which the stomach and the duodenum are found at operation to be invaded by the ulcerative process is now agreed upon by all surgeons. Of 1000 cases of chronic indurative gastric and duodenal ulcer operated on by Wm. J. Mayo, 428 were gastric, 572 were duodenal. Mayo's later statistics show that duodenal ulcers are really more frequent than this, for in 621 cases operated on between June 1, 1906, and January 17, 1911, 32.5 per cent. were gastric, and 64.5 per cent. were duodenal, while in 3 per cent. one or more ulcers of both stomach and duodenum were encountered.

It cannot, however, be argued that these figures are correct for all ulcers. *The figures just given hold good only for the location of such chronic unhealed ulcers that come to operation.* Acute ulcers that heal spontaneously or by medical aid—chronic ulcers in parts of the stomach that may be considered more or less silent areas, and which do not, therefore, run an aggressive course, and chronic ulcers both gastric and duodenal, which are relieved by medical means to such an extent that they do not come under the surgeon's care, are not included in such a table of statistics as that of Mayo's just quoted. How great a number of these non-operative cases there are cannot be computed, nor can it be said that the location of such ulcers is that of those which are demonstrable at operation. *In point of fact, surgical statistics for ulcer are true only as far as they apply to cases seen by the surgeon, but they do not allow of correct conclusions when applied to the various types of ulcer both acute and chronic, aggressive and comparatively quiescent, which come under the care of the internist.*

Position of Ulcers.—Location of Gastric Ulcers.—*Chronic Ulcer.*—Eighty-five per cent. of chronic gastric ulcers involve the lesser

curvature near the pylorus and extend downward, both anteriorly and posteriorly, in a manner compared to a saddle. The posterior ulcer is usually the more extensive. The canal of Jónnesco—the name given to the terminal three-fourths of an inch of the pyloric canal—is not frequently involved, because this canal does not take part in the grinding function of the antrum, and is therefore less exposed to mechanical injury. In a small proportion of cases the ulcer has been found at other parts of the stomach, with some apparent predilection for the posterior wall and the neighborhood of the cardiac orifice.

Acute Ulcers.—The acute ulcers are more generally distributed over the surface of the gastric mucosa. This point is well illustrated by the following table of Fenwick's:

	Chronic ulcers.	Acute ulcers.
Pyloric region	53	13
Middle zone	7	14
Near the cardia	10	12
	<hr/> 70	<hr/> 39

Position of Duodenal Ulcers.—According to Mayo, 96 per cent. of duodenal ulcers are found in the upper portion of the duodenum, extending up to or within three-fourths of an inch from the pyloric sphincter. This is in accord with the figures given by Collins and by Perry and Shaw. According to Collins, duodenal ulcers were located as follows:

In first portion	92.4 per cent.
In second portion	5.4 per cent.
In third portion	1.1 per cent.
In fourth portion	1.1 per cent.

Size of Ulcers.—Ulcers vary in size from minute erosions hardly visible to the naked eye up to 1 cm. to 3 or 4 cm. in diameter. Ordinarily the acute ulcers are of the size of a split pea to that of a penny. Occasionally they are much larger than this, especially when several have coalesced, so that large areas of the wall of the stomach are involved in the process.

Chronic ulcers are somewhat larger than the acute, usually ranging from the size of a ten-cent piece to that of a half-dollar. Occasionally chronic ulcers of the stomach attain very large dimensions, so that cases are recorded in which the ulcer has measured more than 6 inches long by 3 inches wide. Peabody has reported one measuring 19 cm. by 10 cm. In exceptional conditions the whole surface of the stomach between the pylorus and the cardia may be occupied by a single ulcer. These large ulcerations are more often due to the coalescence of a

number of separate areas than to the invasive tendency of a single sore.

Duodenal ulcers are usually quite unimportant in size, strikingly so when compared with the pain and discomfort which they cause. They vary in size from that of a split pea to that of a dime. More rarely they are of the size of a quarter-dollar, while in very exceptional instances several inches of the bowel may be involved.

Number.—Ulcer of the stomach is often spoken of as if it were solitary, whereas in many instances multiple ulcerations are found. This should especially be borne in mind when exploring the stomach surgically, for the finding of an ulcer by such a procedure does not relieve the surgeon from the responsibility of making a further search.

Acute ulcers are more apt to be multiple than is the chronic form. The older statisticians did not recognize this fact as clearly as we do now, and in estimating the proportion of cases in which ulcer was solitary, included in their series both acute and chronic ulcers without making any discrimination between them.

Neither was there any attempt apparently made to separate the gastric from the duodenal ulcerations. For these reasons the older figures are quite misleading. Gastric and duodenal ulcers, and the acute and chronic form of each should be separately considered.

Gastric Ulcer.—In 867 cases of gastric ulcer of both acute and chronic forms collected by Fenwick:

1 ulcer was present in	80.50 per cent.
2 ulcers were present in	12.10 per cent.
3 ulcers were present in	3.10 per cent.
4 or more ulcers were present in	4.26 per cent.

When this writer separated, however, the acute from the chronic form, he found that whereas 87 per cent. of the chronic indurative gastric ulcers were solitary, a single sore was found in but 54 per cent. of the acute variety. This tendency of acute non-indurated ulcers to be multiple has been verified by later surgical statistics.

In rare instances the number of acute ulcerations present in the stomach is excessive. Cases have been reported of such multiplicity that it has been almost impossible to count them.

Recent ulcers are occasionally seen on parts of the gastric wall that are in contact with older ulcerations on the opposite wall when the stomach is collapsed. To them the name of contact or kissing ulcer is given.

Duodenal Ulcers.—Ulcers of the duodenum are usually solitary, except the acute form, which shows a tendency toward multiplicity.

In Fenwick's cases, including both acute and chronic forms:

1 ulcer was present in	86 per cent.
2 ulcers were present in	9 per cent.
3 or more ulcers were present in	5 per cent.

If there are many ulcers they are usually crowded together in the first portion of the duodenum, and the contact or kissing form is not infrequently observed.

Multiple Gastric and Duodenal Ulcers.—Ulcers may be found both in the stomach and in the duodenum of the same patient. In Moynihan's earlier cases of operation for duodenal ulcer, a concomitant gastric ulcer was found in one-half of the cases. This writer quotes W. J. Mayo as finding the same proportion in the cases observed by him. Later statistics, however, lead us to believe that the actual number of operative cases which show associated gastric and duodenal ulcer is much less frequent than has been supposed. In 621 cases of gastric and duodenal ulcer operated on by W. J. Mayo, from June 1, 1906, until January 17, 1911, only 3 per cent. showed the presence of one or more ulcers of the stomach and duodenum. It has been surmised that in these cases the gastric ulcer is the primary sore, the pathological sequence being gastric ulcer, hyperchlorhydria, and finally, peptic ulcer in that portion of the duodenum which first suffers the impact of the hyperacid chyme.

Sex in Ulcer.—It was formerly supposed that ulcer was much more common in women than in men. The oldest statistics of Welsh show that 60 per cent. occurred in the female sex.

In Fenwick's series of cases the proportion in females was as three to two. These figures need revision at the present time.

According to Mayo, duodenal ulcer is found 77 times in men to 23 times in women, while in the gastric ulcer the percentage runs nearly even—52 men to 48 women. This increased percentage of men over women in duodenal ulcer is apparently due to the difference in the position of the duodenum in the two sexes. The curve of the first portion of the duodenum in men is higher and more ascending than that in women, so that the alkaline secretions of the duodenum less readily neutralize the acid chyme. These figures, however, apply only to those cases of ulceration which come to operation, and do not include ulcers that are not surgically treated.

Bradshaw¹ has drawn attention to this point. Of 77 cases of acute gastric ulcer, 72.5 per cent. were women. Of his perforations, 56 in number, 71.5 per cent. were females. Of his cases that came to opera-

¹ Lancet, August 20, 1910.

tion, the proportion between the two sexes was nearly equal, 89 women and 84 men. In the writer's private practice, 40 per cent. of acute ulcers and 68 per cent. of chronic ulcers occurred in men.

It may therefore be stated that acute ulcer of the stomach is three times as common in women as in men, that in chronic ulcers of the stomach the proportion between the two sexes is equal, while in chronic ulcer of the duodenum, three-fourths of the cases occur in men, owing to the anatomical peculiarities of the first portion of the duodenum in the masculine sex.

Age in Ulcer.—In most autopsies it is not definitely stated whether the ulcer is open, healing, or healed, nor is it always possible from the clinical history of the patient to decide at what age it first made its appearance. In 607 cases of open ulcer found by Welsh there were:

Under 20 years	5.4 per cent.
Between 20 and 40 years	37.2 per cent.
Between 40 and 60 years	36.5 per cent.
Over 60 years	20.7 per cent.

It has been supposed that children are but rarely affected. This may be true as to gastric ulcers, but duodenal ulceration in infants is not uncommonly encountered. Chvostek, in 87 autopsies on children under the age of ten years, found duodenal ulceration in 5. Of these infants 1 was three hours old, 1 was four days old, and 1 seven weeks old.

Collins appears to consider the disease comparatively common in childhood, as he finds in 279 reported cases, 42 under ten years of age, and of these 17 occurred in the first year of life. Duodenal ulcer is undoubtedly a cause for melæna neonatorum, and Moynihan has reported 16 cases of melæna in the first week of life, in which duodenal ulcer was found at autopsy.

The cause for these infantile cases is probably the devitalization of a certain area of the gastric or duodenal wall by emboli that originate from thrombosis of the umbilical vein.

It may be said that acute ulcerations, both gastric and duodenal, are more common in the second and third decade, while in the chronic form the symptoms are more apt to appear somewhat later, in the third and fourth decade.

In 100 cases of the writer's, including both gastric and duodenal ulcers, there were:

Between 10 and 20 years	2 cases
Between 20 and 30 years	18 cases
Between 30 and 40 years	36 cases
Between 40 and 50 years	30 cases
Between 50 and 60 years	8 cases
Between 60 and 70 years	6 cases

Etiology.—As early as 1855 Virchow announced that whenever the nutrition of a small area of the gastric mucosa was seriously impaired, chiefly through interference with its blood supply, the peptic powers of the gastric juice produced, by self-digestion, an erosion of this area, resulting in the formation of a gastric ulcer. Since this time our knowledge of the pathogenesis of ulcer has not materially increased. We still believe that in the formation of gastric ulcer, two essentials are necessary, an area of diminished local resistance due to some nutritive or vascular change, and the digestion of this devitalized area by gastric juice. It has been found, however, that some ulcers experimentally produced heal rapidly, while others remain open and become chronic.

It will facilitate the discussion of the subject if we consider:

1. The causes for the local diminished resistance in the gastric mucosa.
2. Conditions influencing self-digestion of this area.
3. Conditions which hinder the rapid healing of the ulcer.

Causes for the Local Diminished Resistance in the Gastric Mucosa.—The chief causes for the lowering of the vitality of a local area of the gastric mucosa are chiefly of a vascular character, embolism, thrombosis of the terminals of the gastric arteries, spasm of the arterial wall producing local anemia, and rupture of the vessel wall, allowing small submucous hemorrhages and hematomas.

Occlusion of a Nutrient Vessel.—That ulcers may follow embolism of the smaller branches of the gastric arteries has been experimentally proved by Panum, who produced gastric ulcer by injecting wax emulsion in the femoral artery of dogs, while Cohnheim has been successful in inducing the same lesions by the introduction of finely powdered chromate of lead into the coronary artery. The oval oblique shape of many gastric ulcers seem to corroborate this theory.

Clinically, this embolic ulcer is seen with special frequency in the gastric ulcerations of newly born infants, the source for the embolism being the clot in the umbilical vein.

Aside from these cases in infants, and cases in which emboli arise from disease of one of the large vessels in the neighborhood of the stomach, as in aneurysm of the celiac axis, embolism of the stomach is exceedingly rare, and can hardly be regarded as one of the usual causes for ulcer. Of 110 fatal cases of malignant endocarditis examined at the London Hospital, embolism in various viscera occurred in 62 per cent. In not a single instance was the stomach affected.

Fenwick found that whenever artificial emboli were thrown into the general circulation of animals, only 3 to 5 per cent. found their way into the gastric arteries, and of these two-thirds occupied the middle

and cardiac zones. In no instance was the pyloric region or duodenum alone affected.

Thrombosis and endarteritis may be causative factors. These are not infrequently the cause for ulcers complicating carcinoma. Rochemont records the case of a perforating ulcer, owing to thrombosis, extending from the base of a neighboring cancer mass. Superficial ulcers are not uncommon in advanced phthisis of thrombotic origin due to lardaceous diseases of the arteries.

Gradual obliteration of a nutrient artery in chronic ulcer is one of the causes preventing cicatrization.

In Howard's cases 48.8 per cent. had arterial sclerosis, and in 22 per cent. the condition was well advanced. Arterial sclerosis is, however, extremely common in the hospital cases from which Howard drew his statistics, whereas ulcer is rare. The causal reaction of arterial sclerosis to gastric ulcer has not been definitely proved, inasmuch as the gastric arteries are not terminal, but end in a large capillary network allowing of free anastomosis.

It is thought that in prolonged pyloric spasm the pressure on the local artery wall, passing as it does obliquely through the muscular tissue before it reaches the mucous membrane, might be sufficient to temporarily shut off the blood supply and allow of the devitalization of an area of the mucosa in that neighborhood. That such a theory is not impossible is borne out by the experiments of Talma, who produced ulcers at the pylorus of animals by faradization of the left vagus at a time when the stomach was distended by food, thus increasing the tension on the stomach wall. Direct pressure on the stomach may be followed by local necrosis and ulceration. In the London Hospital records are described two cases of ulcer due to the adhesion of the sac of an abdominal aneurysm to the posterior aspect of the stomach producing a local point of pressure. Cases have been recorded of ulcer from necrosis consequent upon the pressure of a distended gall-bladder. Inasmuch as the gastric veins have no valves, and as they form an elaborate anastomosis over the entire organ, localized venous congestion is practically impossible. It is improbable that gastric ulcer can therefore arise from any interference with the return flow of blood in the veins.

Hemorrhagic Erosions and Submucous Hemorrhages.—Hemorrhagic erosions and submucous hemorrhages are common in those diseases of heart and lungs which lead to venous congestion of the abdominal viscera, in various cachectic conditions seen in all ages, in acute infections, and in postoperative states, especially after appendix operations. It is from such hemorrhagic erosions that postoperative hematemesis is due. Similar erosions may follow artificial toxemias, such as follow

large doses of diphtheria toxin (Rosenau). These erosions may occur either at the fundus or at the pylorus, may be superficial, or may extend through the entire thickness of the submucosa, show a grayish-red floor and well-defined edges surrounded by a zone of hemorrhagic infiltration. They are small in size, and usually multiple. Healing usually occurs with great rapidity, but they may be regarded as occasional antecedents of ulcer.

Local Injuries.—Local injuries, such as blows in the epigastrium, pressure of mechanics' tools, such as shoemakers' lasts, drilling machines, the carrying of heavy bodies supported in part by resting them against the stomach, and to a lesser extent the continual stooping at work, so that the corsets impinge upon the epigastrium, as in the case of seamstresses, have all been undoubted factors in producing gastric ulcers. These traumatic cases are not infrequent, and Ackermann, writing from Paul Cohnheim's clinic, has described a number of such cases. Ritter, Vanni, and Gross have all produced ulcers by blows on the stomach—the blow causing submucous hemorrhage or hematoma, which subsequently becomes eroded so that an ulcer is eventually produced.

Toxemic Theory.—The toxemic theory of the formation of ulcers has received much attention of late, owing chiefly to the work done by Gandy, and later by Hort.

Gandy has shown that in practically all toxemias there are formed minute gastro-intestinal ulcerations, and that practically in all gastro-intestinal ulcerations there is toxemia. Certain ulcers arising in toxemia, as in the case of duodenal ulcer following extensive burns, are closely allied to simple ulcer in their acute formation, and in their tendency to hemorrhage and perforation. Their earliest stage is ecchymosis, followed by sloughing, hemorrhagic erosion, true ulceration, with hemorrhagic borders, until finally we have either a perforating ulcer, a chronic ulcer with thickened walls, or cicatrization. Gandy has found all of these forms in burns, in infantile diseases, and in various infections including erysipelas, septicemia, pyemia, puerperal disease, infections of the genito-urinary organs in both sexes, pneumonia, typhoid, and a variety of similar diseases. In a number of these infections Gandy was fortunate enough to find instances of each step of ulcer formation. Roberts has collected 16 cases of ulcer following operations upon the urinary bladder, which seems to corroborate this theory.

Türck produced ulcers on dogs by feeding them with cultures of *Bacilli coli communis* for a variable period of time. No healing was observed while the feeding with bacilli continued, but if the bacillus diet was stopped, cicatrization rapidly took place. All his cases showed

signs of severe toxemia, such as hemolysis and parenchymatous changes in the viscera. These changes were similar to those produced by Rosenau in animals used for the purpose of standardizing diphtheria antitoxin.

Hort, who is an exponent of the toxemic theory, believes that in certain toxemias there are formed a hemorrhagin which allows leakage of blood from the arterial wall, and a mucolysin which destroys the mucosa.

The hemorrhagins produce local areas of ecchymosis, and when the mucolysins are formed, an ulcer is developed unless antibodies are elaborated.

Weinland's view is that the stomach secretes an antipepsin or an antibody which prevents self-digestion, and that whenever this antipepsin ceases to be secreted from a certain area of stomach wall, there is nothing to prevent this area from being eroded by its own gastric juice.

Unfortunately, detailed proofs of Hort's and Weinland's theories are lacking.

A very interesting series of experiments on autodigestion have been made by Katzenstein.¹ This experimenter introduced a loop of jejunum with its blood supply intact into an animal's stomach, and retained it in position. On the third day the animal sickened, and died on the eighth day. Autopsy showed the jejunal loop was completely digested. A similar result was obtained by the introduction of the spleen into the stomach through an opening in its wall. A loop of duodenum introduced in a similar way, with its nutritional vessels preserved, was found on the fourteenth day totally unaltered. A portion of the stomach wall itself was inverted in such a manner as to be practically a foreign body lying within the stomach. Eleven days afterward it was found totally intact. His conclusions are that living tissue normally nourished is digested by the gastric juice in the stomach of the same animal, with the exception that tissues producing gastric juice, or normally bathed in gastric juice, when submitted in the same conditions, are not in any way affected.

Whether or not Katzenstein's experiments are accurate may be doubted, for Reering was able in dogs to introduce a loop of transverse colon into the stomach of the same animal and find it intact, and without trace of ulcers after thirteen months, provided that its nutrition through the mesenteric vessel was well preserved.

Causes Influencing the Digestion (Erosion) of the Devitalized Area.—For self-digestion to occur, a certain amount of peptic power of the

¹ Berlin. klin. Woch., 1908, ii, 1749.

gastric juice is generally considered necessary; in fact it has been believed that the formation of a gastric ulcer was usually preceded by hyperacidity and consequent increase in the peptic power of the gastric contents.

A number of cases have, however, been observed, in which recent ulcers have been found associated with carcinoma, and entire absence of any acidity whatever. These instances are, however, rare and accurate observations on the peptic power of the gastric contents in these reported cases are entirely lacking. It may, however, be affirmed that undoubted cases of acute gastric ulceration occur even in the absence of apparent peptic power of gastric contents. More usually, however, gastric ulcer occurs with normal or excessive amounts of hydrochloric acid, and it has been a disputed point whether the hyperacidity that accompanies ulcer is a causal factor or is, on the other hand, the result of the ulcer. It may be said that hyperacidity with ulcer is less frequent than ordinarily supposed.

Causes Retarding the Healing of Ulcer.—It is a well-known fact that ulcers experimentally produced heal rapidly and run a mild clinical course. It is, moreover, the universal experience that frequently in extracting test breakfasts, small pieces of the mucous membrane of the stomach are caught in the eye of the tube and forcibly torn loose, and that evil results from such a traumatism are extremely rare. The writer does not remember ever having observed any disagreeable results from such injuries, although it must be said that the majority of such evulsions occur with achylia, in which condition ulcers are extremely rare. It is, therefore, plausible that a very large number of acute ulcers run a latent course and heal rapidly and completely. Other ulcers, however, do not tend to heal, but persist and become more or less chronic. These are the ulcers that eventually give symptoms and come under the observation of the physician.

It is, therefore, of the utmost importance to discover the reasons for the persistence of ulcer, as it is along such lines that treatment should be directed.

One of the most important observations on this point was made by Quincke and Daettwyler, who proved experimentally that artificially produced ulcers are much slower in healing in animals previously rendered anemic by venesection than in healthy and plethoric ones.

Fütterer excised small pieces of mucous membrane in animals and then kept them anemic by giving them blood-destroying substances. By doing so he kept the ulcer from healing and reproduced the lesions of chronic gastric ulcer, especially when the excised area was in the pyloric end of the stomach near the lesser curvature. This accords with our clinical observation on the cure of gastric ulceration in man

—anemic and cachectic states regularly antagonize the healing of ulcer, and it is not until the general condition of the patient is improved that healing of the ulcer takes place. It is said that anemic and chlorotic girls are especially liable to gastric ulcer, but it may well be that they are no more liable than others to this complaint, but simply that in them the ulcer does not heal, but remains open and gives rise to symptoms.

Matthes showed that after cutting out a piece of the mucous membrane of the stomach, muscular contraction closed the defect, thus protecting the denuded area from further erosion by the gastric juice. In commenting upon this observation, Block suggested that the absence of heavy folds of mucous membrane in the neighborhood of the pyloric portion of the stomach and the absence of submucous tissue, preventing the closure of the defect, accounted for the prevalence of open ulcers in this region. The formation of adhesions between the base of an ulcer and the neighboring parts, while helpful to repair by placing the ulcer in a condition of comparative rest, may also prevent the base of the ulcer from undergoing the degree of contraction which is necessary to cicatrization.

It may be said that ulcers heal more readily in conditions of low acidity than they do when hyperacidity exists. This fact is the basis of our medical and surgical treatment of ulcer—to lower the acidity by medicine, by diet, and by surgical operations on the stomach, for it is generally acknowledged that an excessive acidity not only irritates an open sore and keeps it inflamed, but by increasing the digestive activity of the secretion, it helps to enlarge the size of the ulcer. The writer firmly believes that the persistency of pyloric ulcers is largely due to the hyperacidity which accompanies so large a proportion of these cases, while ulcers elsewhere, that are not attended by such a liability to hyperacidity, are more amenable to treatment and more easily healed.

Another reason for this non-healing of ulcers in the pyloric region is undoubtedly the violent muscular action of the tissues on which they are superimposed.

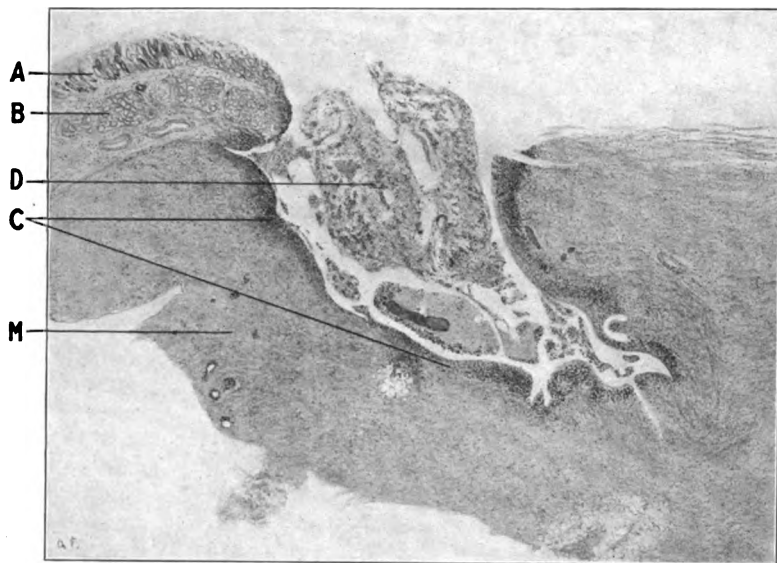
To sum up: The preventable causes for the chronicity of gastric ulcers are:

1. Anemic and run-down condition of patient.
2. Hyperacidity, and in many instances, of pyloric implantation, hypersecretion as well.
3. Overmuscular action of the pyloric portion of the stomach.

Pathology.—The pathology of acute ulcer differs materially from that of the chronic form, so that a separate description is required of each.

Acute Gastric or Mucous Ulcer.—Acute gastric or mucous ulcer cannot usually be identified from the outside of the stomach, and it is often with great difficulty that it can be located, even after the stomach has been opened. The depth of the ulceration depends upon the intensity of the erosive process, and may extend only one-half way through the mucous coat, or may involve the outer coats, and even extend through the peritoneal covering to form a perforation. Its base, therefore, is formed of whatever structure of the stomach happens to be at the limitation of the eroding process.

FIG. 19



Acute gastric ulcer. It has a typical funnel shape and extends deeply into the muscularis *M*. It is partially filled with debris, *D*. To the left is seen the overhanging mucosa, *A*, and submucosa, *B*. To the right these coats are missing. There is intense round-cell infiltration (*C, C*) in the borders of the ulcer.

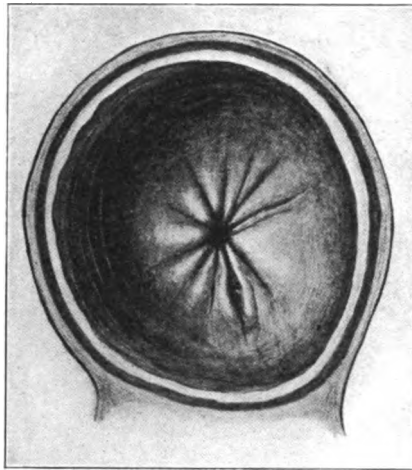
The ulcer is usually round, occasionally oval, and often appears as though it were punched out of the wall of the stomach. The edges are clean and smooth, and are not thickened. The floor is smooth, sometimes necrotic. If the ulcer extends through several of the coats of the stomach it may present a funnel-shaped appearance, as each successive layer of the stomach is usually involved to a less extent than the preceding one.

Microscopically, infiltration with leukocytes or lymphocytes, or both, may be seen between the tubules of the adjacent otherwise healthy mucous membrane, and the base of the ulcer is similarly

infiltrated and necrotic. In the acute ulceration, adhesions to the neighboring part are not ordinarily found, even though the ulcer extend to the serous coat, or even perforate. Lymphangitis is often present, radiating from the base of the ulcer.

The exulceratio simplex, described by Dieulafoy, is practically an acute mucous ulcer, broad and shallow, extending partially or completely through the mucous coat of the stomach. It is often the cause for sudden hematemesis, but the bleeding point is often with great difficulty discovered, even after the stomach has been opened. Being only a type of acute ulcer, it will require no further description.

FIG. 20



Pyloric sphincter seen from the duodenal side, showing duodenal ulcer infolded, resembling fissure in ano. (Codman.)

Codman¹ has made an interesting comparison between ulcer of the duodenum and fissure of the anus. Ulcer of the duodenum usually occurs outside the pyloric ring, so that when the pylorus is contracted the ulcer lies in the mucous folds just as a fissure of the anus lies in the folds of the anal sphincter, and about its periphery there is often a certain degree of inflammatory induration. This infolding of the ulcer, by the mucous puckering during pyloric closure, explains the freedom from pain that occurs when the stomach is full and the pyloric orifice is closed, and also gives us an explanation of the pylorospasm, and of the acute exacerbations which are characteristic of its clinical course. The relation of the ulcer to the pyloric sphincter may explain the difficulty of healing, which is so characteristic of duodenal ulcera-

¹ Boston Medical and Surgical Journal, September 2, 1909.

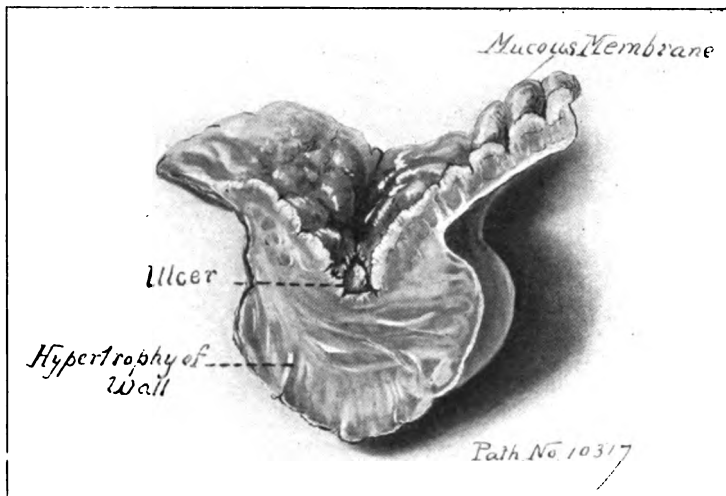
tions, because they are constantly opened and folded in again by the action of the pyloric sphincter.

During the acute exacerbations the thickening caused by inflammatory induration may keep the raw surface from being infolded.

In other cases a round pyloric ulcer may spread, first encircling the lumen of the gut so as to form an annular ulceration, which later progresses toward the stomach.

Chronic Ulcers.—In chronic ulceration we find an attempt at repair, as is shown by the deposit of dense connective tissue in the base and walls of the sore. It would seem that the initial erosion of the mucosa

FIG. 21

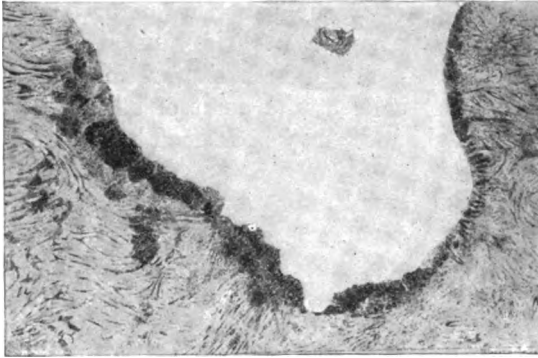


Cross-section of a small gastric ulcer showing thickening of stomach wall at the base. (From Bloodgood's collection of specimens from the Surgical Pathological Laboratory of the Johns Hopkins University.)

by the necrotic process was sufficiently slow for the formation of a barrier of scar tissue to prevent further extension. This connective-tissue barrier may further undergo necrosis, thus gradually increasing the depth of the ulcer. The base may be smooth, or rough and necrotic, especially after an acute attack in which inflammatory symptoms were present. The base is composed of dense connective tissue, infiltrating whatever coat of the stomach has finally resisted the assault of the necrotic process. Thus in shallow ulcers the base may consist of the terminal ends of the glands and of infiltrated interglandular stroma. The muscularis is often greatly hypertrophied. In instances of deeper ulceration the base may be composed of the inflamed submucosa, muscular, or even the subserous coat.

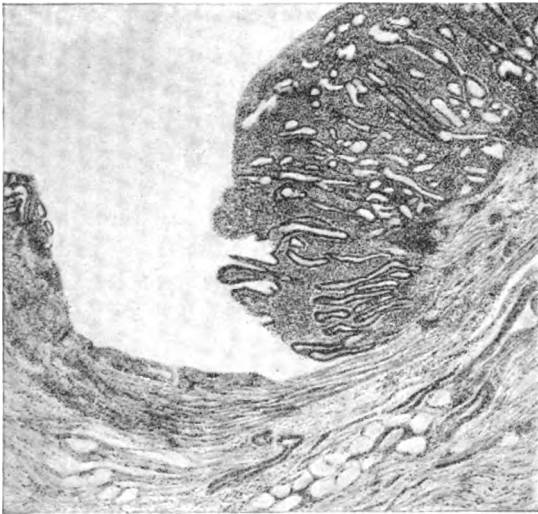
The adjacent mucosa may or may not show hypertrophy. In the larger ulcerations hypertrophy is apt to occur, and is shown by the

FIG. 22



Section through an ulcer with an extensively infiltrated base. (Wilson and McCarthy.)

FIG. 23



Section through the border of the ulcer showing the overhanging hypertrophic border. (Wilson and McCarthy.)

projection of the mucosa over the edge of the ulcer. At no portion, however, of the border can there be seen any trace of epithelial cells dipping below the mucosa into the submucous coat. Lymphangitis is

frequently found radiating from the points of the affected area. Lesions of chronic gastritis are usually found in the neighborhood of the sore.

The inflammatory hyperplasia at the base may extend to and involve the serous coat, and thus may lead to the adhesion of the ulcer to the neighboring parts, such as the pancreas, liver, or abdominal wall. These adhesions are conservative in their nature, and tend to strengthen the weakened points so as to prevent perforation. In course of time, however, the fibrous tissue at the point of adhesion may disappear, so that the floor of the ulcer may be formed by the neighboring organ itself.

In many cases which, to all appearances, seem to be simple indurative ulcers, indubitable evidences of malignancy are present. The fixed and invariable rule should therefore be that every excised ulcer should be carefully examined in every part of its structure for epithelial elements.

Chronic ulcerations are almost invariably distinctly visible by inspection of the stomach or duodenum at the time of an exploratory operation. They can be readily seen and felt. To this rule there are few if any exceptions.

Mansell Moullin¹ has, however, made the statement that ulceration of the duodenum (he does not specifically mention the stomach) may occur without any visible change, post mortem at least, being visible on the serous coat. It is, however, probable that if such cases were examined in the living subject, there would have been at least some local redness to arouse suspicion.

Mayo has drawn attention to the fact that when the pylorus is pulled up for inspection, the traction may interfere with the blood supply, and the local anemia thus produced will cause a white spot to appear on the duodenum just below the pylorus. This may be easily mistaken for duodenal ulcer.

The pathology of the complications, such as adhesions, perforations, and hemorrhages, will be considered under these respective headings.

Healing of the Ulcer.—1. The more acute the ulcer the more readily does it heal and the less evidence is left of its presence. Small acute ulcers may heal so that the scars left are so slight as to be unnoticeable. Deeper ulcers may leave a smooth, white spot where the stomach wall is a little thin and uncovered by normal mucous membrane. Larger and deeper scars may, in healing, so contract as to form puckers, which if sufficiently extensive, may produce various and manifest deformities of the stomach. Such cicatricial contraction in the neighborhood of

¹ Lancet, March 2, 1912, p. 563.

the pylorus readily induces a pyloric stenosis with dilatation of the stomach.

2. Duodenal ulcers may heal leaving simply a deposit of cicatricial tissue in the deep layers, and the mucous membrane is replaced, glands and all. The only way to detect that there has been an ulcer is a gap in which the muscular layer is interrupted instead of being continuous.

3. The ulcer may cicatrize partially, leaving a thickened, dense, connective-tissue mass, which contains but a few bloodvessels, and which shows no tendency to heal. This is the type of ulcer which is not amenable to medical treatment, and which comes most frequently under the observation of the surgeon.

4. The ulcer may undergo malignant degeneration.

ACUTE GASTRIC ULCER

This is often spoken of as "acute perforating ulcer," owing to the frequency of this special complication, and as "medical ulcer," because it is conceded even by the surgeons that the acute forms of gastric ulceration are better treated by medical means than by surgical. The pathological characteristics of the acute ulcer have been elsewhere described. Its complications and sequelæ will be described with those of the chronic form.

Symptoms.—The acute ulcer is characterized clinically by the frequency in which it runs a latent course, and by its greater tendency to hemorrhage and perforation. It is probable that in a very large percentage of cases the ulcer heals quickly without symptoms, or without symptoms sufficiently definite to allow of any diagnosis. The patient complains, if he complain at all, of a temporary distress after meals, a sense of fulness, or the feeling of a load or lump, but not intense enough to necessitate medical advice. It is only if severer symptoms or complications should ensue that the correct diagnosis is made.

Pain.—The most characteristic symptom is pain. According to Fenwick this was the first symptom noted in 21 per cent. of his cases, and of these the pain was severe in only one-third, while in two-thirds the sensation was more like the ordinary distress of indigestion, so that in his series of cases only 7 per cent. began with the characteristic pain ordinarily ascribed to acute gastric ulcer. In the writer's cases, 80 per cent. gave pain as the initial symptom, and of these three-fourths of the cases described the pain as well-marked and fairly characteristic.

The pain varies from a burning or gnawing sensation to a feeling

of soreness in the epigastrium or a painful sense of lump or oppression. It is usually located in the epigastrium, more rarely in one or the other hypochondriac regions, with a tendency to run to the back. In some instances, usually with ulcers of the posterior wall, the pain is felt more severely in the back, or may be felt in the back alone. The pain may occur within fifteen to twenty minutes after the ingestion of food, or it may be deferred until one or two hours after eating. A gastric ulcer may remain painless unless complicated by lymphangitis, in which case the pain is occasioned by normal peristalsis. In this way pain of acute ulcer or acute exacerbation of chronic ulcer may occur soon after eating and continue until the stomach becomes empty.

The duration varies. It may last but a short time, or may continue until the stomach empties itself. In some cases, usually of ulcers at or near the pylorus, with pyloric spasm and hypersecretion, it may be more or less continuous.

Relief from pain occurs in almost all cases after emesis—a fact so well known to the patients themselves that they induce vomiting for the relief it affords. In many cases the relief is complete until the next meal, while in other cases the relief is but partial. Alkaline drinks and soda tablets may give relief, especially if there be hypersecretion. Powders of anesthesine and orthoform (ãã gr. x) will often afford complete relief. This fact is frequently of advantage in diagnosis, as it may be said that epigastric pain promptly relieved by anesthesin and orthoform indicates a lesion of the gastric mucosa. The anesthetic powder does no good, however, in cases of hypersecretion unless combined with sufficient alkali to neutralize the excess of acid.

The effect of diet on the pain is usually well-marked—the coarser the food, the greater the severity of the pain. The beneficial effect of a milk diet is universally acknowledged.

In some cases food taken during a period of pain aggravates it, but in the majority of cases, the ingestion of food is followed by a certain degree of relief.

In rare instances the pain is increased by exercise to a marked degree. This pain on exertion is usually due to an extension to the peritoneum, and a local adhesive peritonitis, and may precede perforation.

Generally speaking, pain in acute gastric ulcer falls into two classes, according to whether the ulcer is at or near the pylorus, *whether on the stomach or duodenal side*, or is at a part of the stomach away from this orifice.

Ulcers at or Near the Pylorus, Gastric or Duodenal.—The pain is of two kinds. In one variety the pain is of a burning, sore, or gnawing character, coming one to three hours after eating, when the food is

passing through the pylorus, and is due to the local irritation of the denuded floor of the ulcer. This form of pain is regularly relieved by eating. Emesis affords relief in proportion to the thoroughness of the act, the vomitus consisting of recently ingested food, often diluted by acid fluid. Anesthesin and orthoform usually afford marked relief at the time of pain. This form of pain occurs in but 35 per cent. of the acute cases, as against 75 per cent. of the chronic ulcers.

The second variety of pain in ulcers at or near the pylorus is of a burning, gnawing character, coming about one hour after taking food, and lasting until the stomach is emptied, either by the natural passage of chyme through the pylorus, or by emesis. Eating usually gives but slight temporary relief. Anesthesin and orthoform are without any marked effect. Temporary relief of a greater or less degree follows the administration of alkalies, granted that these are given in full doses. This form of pain occurs in 30 per cent. of the acute cases.

In these latter cases there is pyloric spasm, with food retention and hypersecretion, and it is the hypersecretion that occasions the pain. The vomited matters are liquid in character, acid in reaction, often excessively so, and contain food remains that have been retained in the stomach for an abnormal period. The vomitus is often of a brownish color dependent upon altered blood.

In many of these cases the pain is not severe, but is described as a "lump," or "oppression," or "distress," coming after meals and lasting more or less continuously until the stomach becomes empty.

A typical instance is the following:

G. W., aged forty-three years. For five days he had complained of a sense of weight and oppression in the stomach, "like a lump," coming one hour after meals and lasting more or less continuously throughout the day, though temporarily relieved by eating. Physical examination: Marked epigastric and dorsal points of tenderness. Vomited matters (induced for relief) four hours after eating are copious, composed five-sixths of liquid and one-sixth of food remains. Total acidity, 54; free hydrochloric acid, 12. Blood strongly positive.

It is quite characteristic of these cases that the most marked distress occurs during the early part of the night. The patient will eat his dinner, for example, at seven o'clock, and after getting in bed will complain of weight and oppression amounting finally to a burning pain, and obtains relief only after vomiting.

The differential diagnosis between acute gastric ulcer at or near the pylorus and ulcer in the duodenum cannot be positively made. Neither can acute gastric ulceration be differentiated from acute exacerbation of a chronic ulcer that hitherto has run more or less a quiescent course.

Ulcers Not Involving the Pylorus.—These accord more with the older descriptions of gastric ulcer in the text-books. The pain occurs soon after eating, generally between one-quarter and one-half hour. Pain coming at once after eating is usually of esophageal origin. Pyloric spasm and hypersecretion do not occur, so that the pain is not so continuous, or it is so much influenced by the administration of alkalies. It is regularly relieved by the natural or artificial emptying of the stomach, and by anesthesin and orthoform. Eating during the period of pain almost regularly aggravates it. This form of pain occurred in 16 per cent. of the acute cases observed by the writer.

Nausea.—Nausea in gastric ulcer is but rarely complained of, and the appetite is generally maintained throughout the disease, although many of the patients are afraid to eat because of the succeeding distress so caused.

Vomiting.—Vomiting is commonly observed, more frequently induced than spontaneous. The characteristics of the vomited matters have been as above described, and may be summed up by saying that in ulcer at or near the pylorus, vomiting occurs because of the hypersecretion, while in ulcers not involving the pylorus, vomiting is for the purpose of ridding the stomach of the food that is irritating the ulcer.

The above description of the symptoms of acute ulcer apply only to those observed in the early stages of the disease, and they will be seen to be somewhat different from those of the chronic form. Should the ulcer pass into the chronic stage, through medical neglect or otherwise, the symptoms approach more and more those of the chronic ulcer, so that it may be impossible to say in any given case whether there is an acute ulcer becoming chronic, or a chronic ulcer during a period of exacerbation.

Hemorrhage, perforation, and other complications of acute ulcer are described, together with those of the chronic form.

Physical Signs.—Physical signs of acute ulcer are usually more evident than in the chronic forms—the epigastric and dorsal points and hyperesthetic zones being especially well-marked. Detailed description of these physical signs are given later (see p. 139).

Diagnosis.—**Gastric Analysis.**—It is a fundamental rule that a tube should never be passed if acute ulcer of the stomach is suspected, owing to the danger of hemorrhage or perforation. For this reason there are few data obtainable as to gastric secretions in acute ulcer. It is only in the atypical cases in which gastric analyses are made before the diagnosis of acute ulcer is suspected that we have any knowledge of conditions of acidity and secretion. In the majority of cases the test breakfast affords no clue. In a certain number of instances, however, the tube withdraws an excess of liquid secretion more or

less mixed with altered blood and containing usually food remains that have been retained an abnormal time in the stomach. The acidity in these cases is not usually far from normal, and may even be sub-normal, especially if much blood be present (see table, p. 136). These are the cases of acute ulcer near the pylorus, with spasm of that orifice and hypersecretion. In other instances the test breakfast is normal as regards secretion and acidity, although the presence of altered blood may afford a clue to the diagnosis. Much can be learned from an examination of the vomited matters, as may be inferred from what has already been said under the heading of *pain* and *vomiting*. The acidity is rarely increased.

Gastric acidity is, however, influenced by gastric hemorrhage. Of 19 cases examined by Fenwick one month after hematemesis, free hydrochloric acid was absent in 17, while in the other 2 only traces could be found. This condition of low acidity persists until the patient is well over the loss of blood, and it may well be that many of the vague symptoms of indigestion that often follow the cure of acute gastric ulcer are due to this lack of hydrochloric acid in the stomach.

Recurrences.—Recurrences are common. Twenty per cent. of Fenwick's cases were readmitted within three years. These recurrences are to be distinguished from the recrudescences of the chronic form by the fact that in the interval the patients are more completely free from symptoms in the acute than in the chronic form, in which a careful history will bring out more or less gastric discomfort between the exacerbations.

Sequelæ.—Acute ulcer of the stomach usually heals quickly without untoward results, although there is a tendency for the ulcer to become chronic. This tendency is increased by lack of appropriate care and diet during the acute stages. Acute ulcers with hemorrhage seldom pass into the chronic form, because of the enforced medical care necessitated by this alarming symptom.

CHRONIC ULCER OF THE STOMACH

The clinical course of chronic gastric ulcer may be subdivided into four groups:

1. Classical pain type.
2. Irregular pain type.
3. Vomiting type.
4. Hypersecretion type.

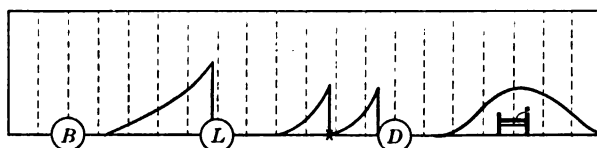
I. Symptoms in Classical Pain Type.—The most definite and characteristic symptom of chronic ulcer is a pain variously described as

burning, boring, drawing, gnawing, cramp-like, or neuralgic, or the feeling of a painful lump or pressure, occurring two to four hours after a meal, and lasting until the patient eats again. It has been described as "hunger pain." This pain was first accurately described by John Abercrombie in 1830, who wrote: "The leading peculiarity of disease of the duodenum, as far as we are at present acquainted with it, seems to be that the food is taken with relish, and the first stage of digestion is not impeded; but that the pain begins about the time when the food is passing out of the stomach, or from two to four hours after a meal."

It is not the kind of pain, or the localization of the pain, that is distinctive, but the *time* of the pain, and the relief afforded by eating.

In many gastric disorders pain appears when the stomach is full—in ulcer, the characteristic pain begins when the stomach begins to empty itself and to force its acid contents through the pyloric orifice. The pain may be referred to the epigastrium, left inguinal region, or to the back, more rarely to one or the other hypochondrium or to the

FIG. 24



Classical ulcer pain. The vertical dotted lines indicate hours. The three circles, B, L, and D, indicate breakfast, lunch, and dinner. The time of retiring is indicated by the outline of the bed. The mark x in this and in succeeding charts indicates extra nourishment.

unbilical region. The location of the pain bears no definite relation to the position of the ulcer. The most that can be said is that in ulceration of the duodenum, pain is often experienced to the right of the median line. This pain in the writer's experience has occurred in 75 per cent. of his ulcer cases. One great characteristic of the pain is its recurrence at a definite time after eating in each patient. The time may not be the same for all patients, but each one has a definite hour at which his pain comes on, and this interval for him is fixed. There is a definite sequence in the time of eating and in the appearance of the pain which is very characteristic.

Another characteristic of the pain is the fact that the larger the meal, the longer the period of relief; pain may occur one or two hours after breakfast and lunch, but is deferred for two or three hours after a hearty dinner.

We must sharply discriminate between distress, uneasiness, or discomfort on the one hand, and actual pain on the other. The former

may occur from a great variety of causes, but the latter, epigastric pain, while occurring occasionally and temporarily as the result of gross diet indiscretions, especially in children, does not recur day after day without an organic cause. *Recurring epigastralgia of a purely functional nature does not occur*—the lesion may not be in the stomach, it may be an appendix or a gall-bladder, but there is a lesion somewhere. A “gone feeling” in the fasting state is not uncommon as a neurosis, but actual pain at this time comes only from an organic cause. The “painful sense of stomach emptiness” or the gastralgokenosis of Boas does not exist as a pure neurosis. A great deal has been written about the pain of hyperacidity. There is no evidence to prove that the intact mucous membrane of the stomach is at all sensitive to acid stimulation—in fact from experimental and clinical evidence it seems fair to assume that hyperacidity as such, does not produce painful sensations in the human stomach, even if there be abrasions of the mucous membrane.

The history of pain of ulcer may extend over years, either steadily, or more usually with periods of comparative freedom. A close cross-examination will, however, nearly always bring out the fact that even in these remissions, minor symptoms are present and the patient's stomach never feels entirely comfortable.

Recurrences of gastric ulcer are most apt to occur during the cold, winter months. In summer the symptoms are almost regularly milder in severity, or even absent.

There are milder cases of ulcer in which the sensation does not amount to actual pain, but to discomfort or uneasiness appearing two to four hours after meals. If we have a patient without gastropotosis, gall-bladder disease, or chronic appendicitis, who complains of constantly recurring distress two or three hours after meals, relieved by eating, we may reasonably affirm the presence of gastric or duodenal ulcer, especially if continuous or alimentary hypersecretion be present.

The quality of food, while making a great difference in the intensity of the pain of acute ulcer, seems to exert very little difference in that of the chronic variety. It is otherwise with the quantity of the food—the larger the meal the longer the period of freedom from pain or distress that follows. Dinner is the most comfortable meal, the middle of the night the most distressing time. The writer has found that the pain seems to depend upon the degree of gastric acidity and hypersecretion, and after analyzing the results of the examination of fasting stomachs and test breakfasts in chronic ulcer, has arrived at the following conclusions:

1. Those patients with ulcer who have the classical pain, have high acidity and an alimentary hypersecretion.

2. Those with "discomfort" have less acidity and less alimentary hypersecretion.

3. Those with slight and irregular pains are apt to have neither high acidity nor alimentary hypersecretion.

4. In those patients with undoubted ulcer who have severe pains, with an acidity not above the normal, complications, such as adhesions, are apt to be present.

It seems to the writer that the pain of ulcer depends upon the excessive amount of gastric juice of high acidity, and not directly upon the nature of the food itself. It is upon the reduction of this high acidity that the whole medical treatment depends.

According to Moynihan, it is a simple thing to differentiate a gastric ulcer from one in the duodenum: If pain occur one or two hours after meals, the ulcer is gastric; if pain occur three or four hours after meals, the ulcer is duodenal; if pain occur one or two hours after meals and then subsides, only to increase in intensity one or two hours later, the patient has two ulcers, one in the stomach and one in the duodenum.

These rules for localization have not seemed accurate in the writer's experience, and he can admit only that pain occurring two to four hours after meals indicates an ulcer *near* the pylorus, either on the gastric or on the duodenal side. A more accurate localization than this does not seem possible.

In ulcer a history of gaseous attacks may be quite as characteristic as the pain, usually running a course parallel to that of the pain, appearing when the ulcer pain is at its height. The greater the acidity the greater is usually the distress from gas. The measures which relieve the pain—food, drink, alkalies, and vomiting—also serve to mitigate the severity of the distention.

Differential Diagnosis of Ulcer Pain.—*Chronic Appendicitis.*—In chronic appendicitis pain referred to the epigastrium may come after food, sometimes earlier and sometimes later, but usually without the regularity observed in ulcer. In some cases, however, a definite regularity can be observed, the pain coming at a fixed and definite time after meals. It may be relieved temporarily by eating, as is the case with gastric ulcer, and is usually worse after exercise. As it is, on the other hand usually relieved by rest, an ulcer cure with bed treatment, is often followed by marked improvement. Twelve per cent. of the writer's earlier cases of supposed chronic ulcer turned out to be cases of chronic appendicitis with gastric symptoms—they were "improved" after an ulcer cure, but the symptoms ceased only after removal of the appendix. Epigastric tenderness may be observed. Pressure over the appendix is not painful in many of the cases. Hyperacidity and hypersecretion may occur with chronic appendicitis as well as

with ulcer, so that a differential diagnosis, by gastric analysis alone, is not usually possible.

It is of importance to remember that in appendicular dyspepsia hematemesis may occur. In over a dozen cases seen by Moynihan, in which no ulcer was found at operation, but in which a cure was effected by the removal of the appendix, hemorrhage from the stomach had exceeded a pint at one time. The cause for the hematemesis is not well understood, but it is probably due to pore-like erosions. Moynihan's observations, made on these cases of chronic appendicitis giving gastric symptoms, are most interesting. He has noted at the time of operation that from time to time there is a condition of vigorous contraction involving the pylorus and the pyloric antrum, the affected area becoming thickened, contracted, and pale. It would seem as if the pylorus served as a guard to the bowel distal to it, and that in inflammatory and irritative conditions its protective control extended to the whole of the midgut. To show how close may be the mimicry of the two diseases, even to the symptoms of perforation, reference should be made to the clinical history of a case of chronic appendicitis with epigastric pain and perforative symptoms narrated under the heading of Chronic Appendicitis (p. 571).

Arterial Sclerosis.—Arterial sclerosis may give rise to epigastric pain occurring after meals, and may closely simulate that due to ulcer. The pain or, as is more usual, the feeling of discomfort, usually appears earlier than in ulcer, being most marked at the height of gastric activity, when the demand is made for an increased blood supply for the physiological processes of digestion. It is suggestive of the arteriosclerotic origin of the pain that it does not usually occur when the patient sits or lies down after eating, but regularly appears if exercise is indulged in after a full meal. In almost all of these cases flatulence is marked, and relief from the distress follows free eructations. This is rarely observed with ulcer. Anginoid symptoms occurring spontaneously or after exertion are of frequent occurrence, and are of material help in diagnosis.

Diseases of the Gall-bladder.—Diseases of the gall-bladder may give rise to pain closely resembling that of ulcer. The regularity of the pain is, however, not as marked as in ulcer, appearing "at any time," without being influenced by the kind of food that is taken. Marked variations occur from day to day, and there are frequent periods during which the pain is more or less constant, associated with tenderness limited to the gall-bladder region, usually associated with localized rigidity of the right costal arch and of the upper portion of the right rectus. Hyperacidity occurs in only 30 per cent. of cases, many cases showing subacidity or achylia.

The diagnosis is usually made without much difficulty. In other cases no definite opinion can be expressed, and the uncertainty in diagnosis is increased by the fact that cholecystitis and gastric or duodenal ulcer often occur together in the same patient. Adhesions between the gall-bladder and the pyloric region of the stomach may give rise to dragging pains whenever they are stretched by the mechanical presence of food and cause pain or discomfort during the digestive period. These pains are increased by exercise and are relieved by physical rest.

Patients who habitually bolt their food without masticating and especially those with defective teeth, are very apt to suffer from epigastric pain after a meal. The distress occurs only after the taking of solid food, and ceases when liquid food is enjoined, or when the patients follow instructions as to the proper methods of eating.

II. Symptoms in Irregular Pain Type.—Pain in Left Inguinal Region.—A small number of patients, amounting to 5 per cent. of the writer's cases, refer their pain to the left inguinal region, the pain coming two to four hours after meals, relieved by eating and uninfluenced by the condition of the bowels. In two of the cases operation showed the ulcer to be on the posterior wall near the cardiac orifice.

The following is the history of one of these patients:

G. McL., aged forty-two years, for twenty years has suffered from sharp localized pain in the left inguinal region, uninfluenced by defecation, coming two to four hours after eating, and relieved only by eating again. There have been many periods of intermission, but the character of the pain when it occurs is always the same. From time to time he vomits acid scalding fluid. At no time has he ever had pain in his epigastrium.

Physical Examination.—Localized tenderness in epigastrium; no dorsal point. Sigmoid normal. No evidence of renal calculus.

Gastric Analysis.—Fasting stomach: 30 c.c., fluid, with a few starchy remains; total acidity, 30; free hydrochloric acid, 16.

Test Breakfast.—30 c.c., pasty consistency; some gastric mucus. Total acidity, 68; free hydrochloric acid, 34. The pain was so severe and uninfluenced by medical treatment that operative interference was resorted to, and an ulcer the size of a fifty-cent piece was found in the lesser curvature of the posterior wall near the cardia. This was excised.

Recovery was slow, nausea, vomiting, and pain remaining.

A gastro-enterostomy was done four weeks later, and since that time the patient has remained in perfect health, without a trace of pain or discomfort. The classical time at which the pain occurred, and its relief by eating, are interesting and instructive features.

Pain in the Back.—This is frequently associated with the epigastric pain, but it may occur as a solitary point of localization.

(a) It usually appears two or three hours after eating, and is relieved by food. Indicating an ulcer on the posterior wall, physical signs are usually lacking, and there is not apt to be hyperacidity. In the writer's experience this back pain is often a precursor of perforation.

T. H. S., aged twenty-eight years. Patient was practically free from indigestion until two weeks ago, when he began to complain of a disagreeable pressure feeling in the epigastrium which merges gradually into an intense pain in the middle of the back. His symptoms are at once relieved by eating, and do not reappear for several hours after his meal.

Patient was placed on ulcer cure. On tenth day symptoms of sub-acute perforation occurred, complicated by empyema. Recovery slow but eventually complete.

(b) Pain in the back, of a steady, boring character, without the relief afforded by eating, often severe enough to necessitate occasional doses of morphine, suggest adhesions to and probably erosion of the pancreas. Such a case is the following:

M. B., aged forty-three years. Formerly a heavy drinker, but has been abstinent for over ten years. One year ago began to complain of severe pain beginning in the epigastrium and running through to the back, coming three or four hours after eating, and lasting until he ate again. For the past three weeks he has suffered from a steady, boring pain in his back, not influenced by eating. This pain has been so steady and severe that he has been kept more or less under morphine the greater part of the time.

Physical examination shows tenderness to the right of the median line, one inch above the navel.

Gastric Analysis.—Fasting, negative. Test breakfast: 10 c.c., watery, scanty food remains of roll. Total acidity, 30; free hydrochloric acid, 20. Operation shows ulcer on posterior wall, perforating into pancreas, and causing a well-marked erosion of the pancreas at this point.

Exsection, suture of pancreas. Gastro-enterostomy. Perfect restoration to health.

Pain Due to Adhesions.—According to Drummond,¹ pain between 1 and 2 A.M. almost always is due to ulcer that is adherent to the pancreas. This cannot be verified by the writer.

Pain at once after eating, depending for its intensity upon the mechanical weight of the ingested food, suggests perigastric adhesion.

¹ British Medical Journal, July 10, 1909.

Constant pain during the day and diminishing during the latter part of the night is also seen in ulcers of the lesser curvature near the cardiac orifice with adhesions.

Pain in the right shoulder after exercise or after meals indicates adhesion between the ulcer and the liver.

Pain after Ingestion of Food.—There is a type of ulcer near the cardiac end in which pain occurs soon after the ingestion of food, and continues until the stomach empties itself. Hyperacidity and hypersecretion are not present.

Two peculiar features may be observed in these cases: (1) There is a marked intolerance for hot food or drink. The patient will sip a little soup and almost before the hot liquid has had time to reach the stomach will gulp down two or three swallows of iced water with instant relief to the burning. Wines, especially Port, Madeira, and Sherry, will often produce the same instantaneous burning pain. (2) The patient may be perfectly free from all symptoms during the day, but on going to bed at night the pain at once comes on and remains until some alkaline food is drunk, after which there is instant relief. The pain may be brought on again by turning on the left side. There is no pain or discomfort in these cases when the stomach is empty.

It may be that these cases cannot be easily distinguished from gouty hyperesthesia of the throat and esophagus. In the gouty cases the hyperesthesia is diffused, extending from the pharynx downward, the whole passages being sensitive to the insertion of a stomach-tube. Throat symptoms are prominent both subjectively and objectively. The gouty cases are, furthermore, aggravated by ulcer treatment, and improved rather by fresh air and exercise.

Pain during Deglutition.—It may infrequently happen that an ulcer due to peptic erosion involves the lowest segment of the esophagus just above the cardiac orifice. In all characteristics it is the same as if of gastric origin. Pain is noted as occurring during the act of deglutition—the passage of a tube is painful. These symptoms can as well occur in ulcers just distal to the cardia, so that a differential diagnosis may be impossible except by the use of the esophagoscope. This latter method of diagnosis is, however, so dangerous in these cases as to be absolutely unjustifiable.

There are occasionally radiations of the pain in true gastric ulcer, so as to be experienced with greatest intensity at the level of the upper portion of the esophagus, but Ewald, in describing these cases, lays emphasis on the fact that the passage of a tube is not painful, thus rendering improbable the esophageal implication of the lesion. Such a radiation upward of the pain of gastric ulcer has not occurred in

any of the writer's cases. Cardiospasm may follow, even after complete healing of an ulcer of the cardia without cicatricial contraction.

Pain Due to Reverse Peristalsis.—There is a peculiar type of pain referred to the cardia, which may occur even if the lesion be pyloric or duodenal. This pain comes in waves of a few moments' duration, of a distressing character, and gradually recedes, or else is suddenly and completely relieved if gas or sour eructations be raised. These waves of pain recur until the stomach is emptied naturally or by emesis, or until sufficient alkalies are given. The cause for this pain is pyloric spasm, hyperacidity, and hypersecretion, followed by reverse peristalsis.

Constant Gnawing Pain.—Constant gnawing pain uninfluenced by food, coming in attacks, occurs in a fairly large percentage of cases. The attacks may last a few days or a few weeks, and are followed by periods of comparative freedom, although in these free intervals a history of some degree of pain or distress may usually be elicited.

The cause for the constancy of the pain is often difficult to determine, although in the main the cases may be divided into two groups.

In the *first group* the pain is evidently due to adhesions or erosions of adjacent viscera, and usually indicates an acute extension of the ulcer. It often precedes perforation or ulcer. Such a history is as follows:

F. B., aged fifty years, for five years has complained of pain in the left iliac fossa, coming two or three hours after eating, lasting until the next meal. Has had intermissions of two or three months at a time. Has had frequent attacks of constant severe gastric pain, uninfluenced by eating, and necessitating the use of morphine. These attacks last usually two or three weeks, and are not accompanied by nausea or vomiting.

On operation an ulcer was found in the lesser curvature and posterior wall, near the cardia, with extensive adhesions to the pancreas.

It is interesting to note the association of the pain in the left iliac fossa and the location of the ulcer.

In the *second group* are placed those cases of constant pain due to acute exacerbations of a chronic ulcer, especially if near the pylorus, causing a sudden partial closure of the pyloric opening, either from tumefaction or from spasm. As the result of the pyloric closure, acute hypersecretion occurs, and it is from the continual irritation of the ulcer by the continuous acid secretion that the constant pain originates. This pain is often relieved by eating or by taking soda, but the relief thus afforded is incomplete and temporary. More marked relief follows emesis, the vomitus consisting of large quantities of acid fluid,

which may or may not contain blood. In severe cases acetoneuria is present.

These hypersecretion cases will be discussed more in detail under a separate heading.

III. Symptoms in Vomiting Type.—There is a distinct type of chronic ulcer characterized by recurring attacks of nausea and vomiting, with periods of complete freedom. The vomited matters consist only of what has been recently eaten, never of acid fluid or residual food; the stomach simply rejects what has been put into it. Acute gastritis is the diagnosis usually made, but the attacks are more prolonged than in simple gastritis, frequently lasting seven to ten days.

The patient experiences a sense of weight or oppression one-half to two hours after eating, followed by nausea and vomiting. There may be severe retching after the stomach has been emptied. In other cases the vomiting is erratic as to time, and absolutely without relation to the taking of food.

The characteristics then of this group are: (1) Prolonged irritability of the stomach to retained food. (2) Negative character of the vomited matters. These are not sufficiently characteristic to enable one to be positive about the diagnosis, but chronic ulcer may be suspected in any case of recurring vomiting of recently ingested food, lasting longer than three days, that is not due to gall-bladder disease or to appendicitis. At any time more characteristic symptoms of ulcer may appear. Such a history is as follows:

S. S., aged thirty-eight years, has been accustomed to take three to five cups of strong tea a day, and to eat between meals. Alcohol denied. Bowels always constipated. For six years has been subject to occasional attacks of indigestion following indiscretions in diet—weight in the stomach, heart-burn, and the belching of gas. There was no pain or tenderness, and until a year ago there was no nausea or vomiting. One year ago, during one of these attacks, she developed gastric irritability so that for a number of days she was quite unable to retain any food. The vomited matters consisted merely of what she had recently eaten.

From this attack she recovered and for three months was able to eat all sorts of food without discomfort.

Eight months ago she had a similar attack, though more severe, lasting two weeks. She was then well for four months.

Three and a half months ago she again had an acute outbreak; she suffered from frequent attacks of nausea and vomiting, although without any definite relation to the taking of food. She left off one article of diet after another, being finally reduced to zoolak and oat-meal gruel, losing constantly in flesh and strength, and spending most

of her time in bed. Two weeks ago her nausea and vomiting became worse and she has retained very little on her stomach since that time. At no time has there been vomiting of blood, melena, pain or tenderness.

On admission physical examination was entirely negative except for marked atony of the ascending colon.

Gastric Analysis.—Fasting stomach, 50 c.c., greenish fluid, without food remains. Total acidity, 54; free hydrochloric acid, 6. Blood negative. Weak erythrodextrin reaction.

Test Breakfast: 40 c.c., well digested. Total acidity, 90; free hydrochloric acid, 70.

During the first week of treatment she vomited once every day without relation to meals, but never at a time when the stomach should normally be empty. After the first week she was up and around, eating everything and complaining of no gastric discomfort whatever.

Readmission.—She was readmitted to the hospital four weeks later with the following history:

After the patient left the hospital she remained free of distress, though she was quite weak. She ate plain food with relish.

Two weeks ago without any evident cause she was suddenly seized with severe vomiting and intense, stinging pain in the epigastrium. The vomitus was thin and copious. The pain was partially allayed for a time by eating, but ceased entirely only after free emesis. These symptoms had continued for the past two weeks without amelioration.

Physical Examination.—The patient was weak and emaciated, evidently suffering acute pain. There is frequent vomiting of food and acid water without admixture of blood. Abdomen retracted and scaphoid, but is everywhere insensitive. No dorsal point. Lower curvature 4 cm. above the umbilicus.

Examination of Vomitus.—30 ounces of brownish, turbid fluid with a finely divided, brownish black sediment. Blood positive. Total acidity, 80; free hydrochloric acid, 48.

Microscopical examination shows a few undigested starch cells, much brownish granula detritus of altered blood. No sarcinæ or bacilli.

Patient was at once put on the von Leube ulcer treatment with atropine administered hypodermically. In spite of this treatment, however, she continued to have pain and to vomit brown acid fluid giving positive blood reactions. This often amounted to over three pints in the twenty-four hours.

On the twenty-second day of treatment she had a severe hematemesis and passed tarry stools. She was rallied only with extreme difficulty.

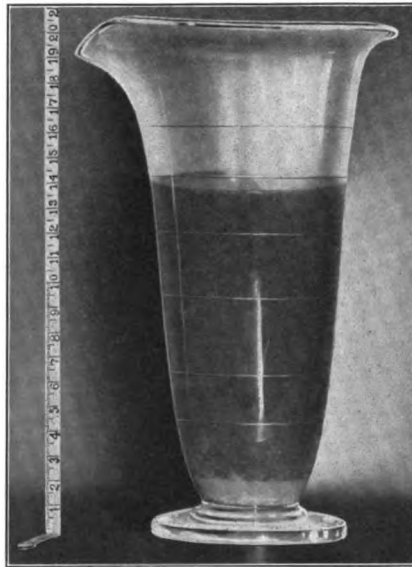
All medical efforts failing to relieve her condition, gastro-enterostomy was performed. The pylorus was found to be much thickened by an

old ulcer causing well-marked pyloric stenosis. The thickness of the ulcer base indicated that the lesion must have existed for months, more probably for years. There were no adhesions.

IV. Symptoms in Hypersecretion Type.—The subject of hypersecretion is elsewhere discussed. Under the present heading will be therefore included only the types of hypersecretion that are clinically associated with the course of gastric ulcer, embracing the acute, chronic, and alimentary forms.

Acute Hypersecretion and Ulcer.—Acute hypersecretion is characterized by attacks in which the gastric juice is poured out in large quantities both in the fasting and in the digesting state.

FIG. 25



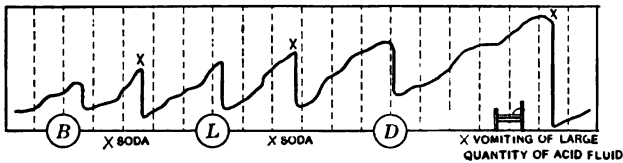
Vomit of acute hypersecretion of ulcer. This represents the amount of fluid aspirated from the stomach after abstinence from all food and fluids for twenty-six hours. Total acidity, 68; free hydrochloric acid, 52. A finely chymified food residue is seen as a sediment.

It may occur as an early symptom of ulcer, or may appear at any time during the course of the disease, even while the patient is undergoing a rigid ulcer cure. Its cause is regularly a sudden pyloric obstruction, most commonly from a muscular spasm of the pyloric ring, and almost regularly implies an ulcer at or near that orifice. It is one of our most reliable means of localization.

There is the complaint of more or less epigastric pain and heart-burn, or else a sense of gastric fullness and nausea. These subjective symptoms are relieved to a very slight degree only, and but for a short

time, by eating or by alkaline drinks. Eating may aggravate the discomfort. Complete relief comes only after thorough vomiting. The act of vomiting is not usually accompanied by much muscular effort, but the fluid easily gushes out, often in large quantities. The vomited fluid is either colorless, slightly green in tinge, or else brownish from the admixture of altered blood. There are no visible remains of ingested food unless food has recently been taken. The acidity is almost invariably high, ranging usually between 90 and 120, the greater part of the acidity being due to free hydrochloric acid.

FIG. 26



Pain curve in acute hypersecretion of ulcer.

There is relief after the vomiting for a time, usually for one to three hours, but distress occurs after this time, merging into pain with a renewal of the vomiting. There is thus a certain periodicity in the symptoms—usually the early night hours are the ones in which the pain is more apt to occur.

Such an attack of hypersecretion may last a few days or it may be continued throughout the disease. Occurring in short attacks it is often mistaken for acute gastritis, but the fact that the vomitus is of acid fluid and not of food, and that it occurs whether the patient eats or not, should definitely exclude acute gastric catarrh. Occurring during the course of ulcer it is usually amenable to medical treatment, although in severe, untractable cases operative interference is often necessary. Operation during the attack is, however, to be deplored, owing to the desiccation of the patient's tissues by the withdrawal of so much liquid from the system. The history of such a patient is as follows:

Mrs. H., aged thirty-five years, was well until one year ago, when she suffered from gnawing pain in the stomach, relieved temporarily by eating or by vomiting, the vomited matters consisting of "acid scalding water," that she would vomit "by gushes." This acid fluid vomiting would continue the same whether she ate or not. After a month the symptoms subsided and she was comparatively well, although she complained of moderate distress two hours after meals, and occasional heart-burn. Two months ago she suffered in like manner from

pain and acid-fluid vomiting, which continued in spite of almost complete abstinence from food for fourteen days, and then subsided, only to reappear five days ago.

Physical examination shows well-marked epigastric and dorsal points. Splashings are readily elicited to the umbilicus, although no food or liquid had been taken for twenty-four hours. There is moderate acetoneuria.

The vomited matters are watery, slightly greenish, and do not contain any food remains.

Total acidity, 104; free hydrochloric acid, 84. Lactic acid, negative. No blood, sarcinae, or bacilli. Estimated quantity for past five days is between two and three pints as a daily average. Liquid nourishment has not exceeded 10 ounces on any day. Under Lenhart's diet and large doses of bismuth subcarbonate, she improved steadily and remained comparatively free from distress for three months, at which time she had a large hemorrhage and a recurrence of her acid vomiting. Operation was proposed and rejected, and the patient passed from observation. One year later it was learned that she was in a sanatorium with the same complaint.

Chronic Hypersecretion in Ulcer.—Chronic hypersecretion consists in the finding of gastric juice both in the fasting stomach and in the digesting stomach. It differs from the acute form not only in its chronicity, but in the fact that it never reaches the excessive degree commonly seen in the acute cases. The theories for its pathogenesis are elsewhere given, but the author believes firmly that continuous secretion means only one thing—mild pyloric stenosis.

In 50 per cent. of the writer's cases of ulcer the stomach contained in the fasting state, 30 to 60 c.c. of pure gastric juice without the admixture of food remains. The symptom to which it gives rise is hyperacidity or heart-burn at any time of the day or night excepting directly after meals. These patients are never without their soda mints, and they keep taking them long after the hour at which the stomach should normally be empty. These cases are often diagnosticated as suffering from "hyperacidity" or "acid catarrh of the stomach."

Chronic hypersecretion is, however, only a presumptive proof, at best, of ulcer. Its chief significance is that it affords the best means available of localization, implying regularly a lesion at or near the pylorus, with some degree of narrowing of that orifice.

Alimentary Hypersecretion with Ulcer.—In alimentary hypersecretion there is an excessive quantity of gastric juice secreted during the digestive period. The fasting stomach is practically empty. It is present in many chronic conditions of the stomach in a mild degree, the upper layer of surplus gastric juice in the test breakfast, after

standing, never exceeding in depth that of the underlying layer of digested breadstuffs.

In ulcer we have a distinct group in which the amount of the supernatant layer of surplus gastric juice is much greater than this. The fasting stomach is empty, so that pyloric stenosis is definitely excluded.

The test breakfast separates on standing into two layers. The upper layer of surplus gastric juice is from four to twenty times the depth of the lower layer of digested breadstuffs. The acidity is almost invariably high, total acidities of 90 to 110 not being uncommon.

Such a condition may give rise to symptoms which may be readily inferred—pain or heart-burn two or three hours after meals, at the height of the hypersecretion, relieved by eating or by vomiting, or by taking soda.

In other cases distress after meals, or the feeling as if the patient had gas on his stomach, when in fact none is there, are the chief symptoms. It differs from the course of ulcer with chronic hypersecretion in that there is no distress at the time when the stomach should be normally empty.

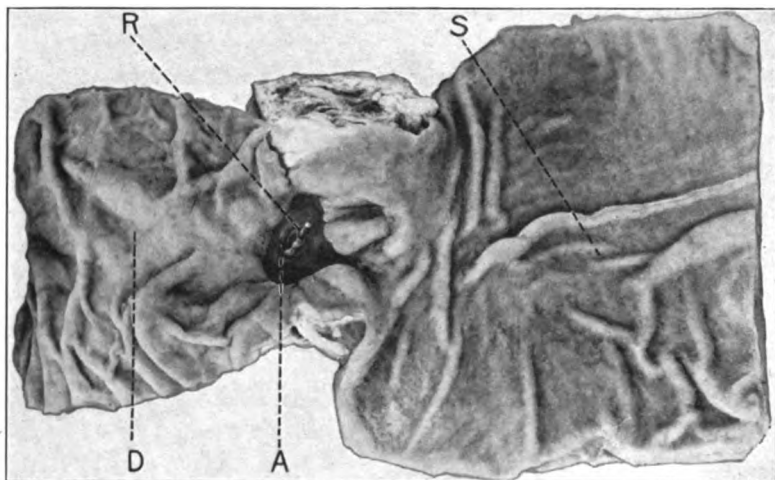
In a large number of these cases a von Leube ulcer cure will relieve the patient from all discomfort, and from his clinical history we would infer that he was again possessed of a normal digestion. The examination of the gastric contents will almost invariably show the same amount of alimentary hypersecretion and of high acidity, the only difference being that the patient feels well. This freedom from subjective discomfort, with the continuance of unchanged conditions of gastric secretions, would seem to indicate that the ulcer had either healed or had become insensitive to acid stimulation. And so these patients continue for years. In some instances the subjective sensations return, while in others the patients feel well and able to digest anything without discomfort, but still show their alimentary hypersecretion and their high acidity. The true nature of the complication is not known at the present time.

HEMORRHAGE IN ULCER

It is probable that the great majority of ulcers give rise to some degree of bleeding at some time during their clinical course. Hemorrhage is, however, only detected when visible blood is present in vomited matters or in the stools, or whenever clinical tests of gastric or intestinal contents reveal the presence of blood, which by processes of exclusion can be inferred to originate from the stomach. We distinguish, therefore, visible hemorrhages and occult hemorrhages.

Types.—Visible Hemorrhages.—Visible hemorrhages in the form of hematemesis or melena occur in a large number of cases of ulcer of the stomach, variously estimated by different observers. 80 per cent. of Lebert's cases were marked by hemorrhage, 71 per cent. of Fenwick's, 50 per cent. of Ewald's. Hemorrhage complicated 80 per cent. of ulcer cases at the University College Hospital, and 60 per cent. of similar cases at the Royal Victoria Hospital. Stockton, on the other hand, finds hemorrhage in only 25 per cent. of cases, and his compiled statistics show that only 28 per cent. of ulcers give rise to visible hemorrhages. Statistics on this point are apt to be very misleading. In hospitals the severer forms of ulcer, especially those with hemorrhage, are relatively more frequent than ulcers which do not give rise to such alarming symptoms.

FIG. 27



Duodenal ulcer showing erosion of an artery in the base, from which fatal hemorrhage occurred.

In the writer's cases 49 per cent. of those in hospital practice gave the history of vomiting of blood, while in his private practice only 25 per cent. gave this history. It is not because the type of ulcer is different, but because those ulcers which do not bleed are apt to be treated in dispensaries, while patients with hemorrhages are alarmed, and naturally enter the hospital wards. Undoubtedly many cases of hemorrhage from esophageal varices are included among the ulcer cases.

The source of the hemorrhage is usually from an eroded artery—less frequently of venous origin.

Savariaud in 54 cases found a venous origin in 4 cases, the splenic artery in 17, coronary artery in 6, gastric artery in 10, pancreatico-

duodenalis in 7, while in 10 the source of the hemorrhage was undetermined. The bleeding may come from the rupture of a small artery at the base of the ulcer, from erosion of the spleen itself, or even from the aorta.

The hemorrhage may occur at any time, but especially after meals during the digestive congestion of the stomach, and at a time when the walls of the stomach are put on a stretch.

In some cases the hemorrhage is preceded by an increase of the ulcer pain, from which we may infer that an extension of the ulcerative process has taken place.

If the hemorrhage be large and sudden, the patient will suddenly become faint and dizzy. There may be pain, often quite severe, preceding the onset of the bleeding, and relieved by the flow of blood. Sudden anemic symptoms develop according to the amount, to a less extent upon the rapidity of the hemorrhage—pallor, faintness, even complete syncope, collapse with dyspnea, amounting to air hunger in the severer cases, rapid pulse of low arterial tension.

The shock may be so great as to inhibit attempts at vomiting, but in the majority of cases blood is vomited, more or less clotted, and more or less altered in color, according to the length of time it has been retained in the stomach. Admixture of blood with food, and blood of a brownish tinge from the action of the gastric juice, are indications of its gastric origin, although it must be remembered that blood from rupture of esophageal varices or even of pulmonary origin may pass into the stomach and thence be vomited.

The reaction from gastric hemorrhage is usually accompanied by moderate fever.

Within a few hours or days, blood appears in the stools. The typical stool under these circumstances is of a tarry appearance and quite offensive. It may be difficult to determine by gross examination whether the blackish color is due to medicine recently administered, such as bismuth or iron, or is due to altered blood, but upon shaking the stool with a little water there is usually a crimson or magenta discoloration of the water which is quite distinctive. Chemical tests for food of course give final and definite information. If the hemorrhage be large, and rapidly passed through the intestines, the color of the blood is brighter, although it may be said, as a general rule, that blood passed per rectum, that is by its color and appearance recognized as blood, rarely comes from a source as high as the stomach. In the cases of melena without hematemesis, the source of the bleeding is usually from a duodenal ulcer.

If the hemorrhage be less marked, and not accompanied by vomiting, there may be only an attack of faintness and a sudden pallor. If the

hemorrhages are repeated, even if moderate in amount, the only symptom may be an increasing anemia. This may be so extreme as to simulate pernicious anemia or malignant cachexia. The rule should be to examine stools for blood in every case of sudden or obscure anemia.

In Acute Ulcer.—Hemorrhage in acute ulcer often occurs as an initial symptom preceding the advent of pain. This occurred in 75.4 per cent. of Fenwick's cases. This seems to the writer to be altogether too high a figure, as in his experience hemorrhage occurred as the first symptom in but 20 per cent. of the acute cases.

The hematemesis of acute ulcer is regularly more sudden and profuse than in the chronic forms of ulceration for the reason that the walls of the ulcer are soft and the vessel wall normal, whereas in the chronic form the walls of the nutrient vessels are often thickened and its lumen diminished.

FATALITY.—The fatality of hemorrhage varies according to different observers.

Rodman claims that 40 per cent. of cases with gastric hemorrhage die; Mayo Robson estimates the fatality at 64 per cent. Fatal hemorrhage, on the other hand, occurred in but 5 per cent. of Brinton's cases, and 3.4 per cent. of Fenwick's. Robson claims that hemorrhage causes the death of 7 per cent. of ulcer cases, basing this figure from a compilation of statistics.

The writer's experience is as follows: In cases taken from both hospital and private practice, hemorrhage, mild and severe, occurred in 37.6 per cent.—fatal hemorrhage in 3.1 per cent. The proportion of non-fatal to fatal hemorrhage is, therefore, as 10 to 1.

It is interesting to note that while the proportion of hemorrhages is much greater in the hospital series, the proportion of hemorrhages that are recovered from to those that are fatal is the same in both series.

	Cases.	Per cent.
Private cases	82	
No hemorrhages in	60	75.0
Hemorrhages in	22	25.0
Fatal in	2	2.5
Proportion of non-fatal to fatal, 10 to 1.		
Hospital cases	77	
No hemorrhages in	39	51.0
Hemorrhages in	38	49.0
Fatal in	3	4.0
Proportion of non-fatal to fatal, 12 to 1.		
Total cases	159	
No hemorrhages in		65.5
Hemorrhages in		34.5
Fatal in		3.1
Proportion of non-fatal to fatal, 10 to 1.		

Melena in infants occurs once in about 1000 live births, but in only a very small number of cases can it be traced to ulcer. The number of infants, however, who die from duodenal ulcer is greater than is ordinarily supposed. The hemorrhage may appear shortly after birth, and be quickly followed by death in collapse. Vomiting of blood is rare: melena being usually the only manifestation of the bleeding.

Helmholz,¹ in a recent interesting article, has brought to our attention the frequent association of duodenal ulcer and marasmus. Of 16 cases of infantile atrophy coming to autopsy, duodenal ulcer was present in 8. The ulcers may be single or multiple, their edges are usually more sharp and abrupt than those found in adults. In none of the cases was there any evidence of repair. In these marasmic cases a long period of weakness, wasting, and anemia preceded the occurrence of hemorrhage. Helmholz believes that the long and wasting disease so enfeebles and devitalizes the infant that it falls an easy victim to the disease.

To the history of these 8 fatal cases Helmholz adds that of the ninth case of marasmus and melena—in all probability due to duodenal ulcer, which eventuated in recovery.

It is often difficult to decide whether hemorrhages are really of gastric origin. Blood may come from lung, nose, or throat, and be swallowed and subsequently vomited, with all the characteristics of hematemesis of gastric origin. In these cases, however, the presence of blood-stained sputum or the examination of nose and nasopharynx usually reveals the initial set of hemorrhage.

Blood of esophageal origin may be more difficult of detection.

Most common are the esophageal varices consequent on the venous stasis of cirrhosis of the liver. In these cases the previous history is rather that of alcoholic gastritis than of ulcer. Hyperacidity symptoms are but rarely observed, as subacidity or even achylia is the rule, while the physical symptoms are those of hepatic cirrhosis. It must be remembered, however, that cirrhosis often occurs without changes in the size of the liver, and that enlargements of the spleen may disappear after the reduction of the portal congestion by free bleeding. As a rule, hemorrhage from esophageal varix appears suddenly and profusely, but is not apt to be repeated in that patient. Repeated hemorrhages suggest gastric ulcer.

Esophageal bleeding may occur from the gradual leakage or sudden perforation of a thoracic aneurysm. The previous history should clear the diagnosis beyond point of doubt. Patients, however, who enter the hospital with such an accident usually die before a full clinical history can be obtained.

¹ Deutsch. med. Woch., 1909, i, 534.

Hemorrhage from the stomach occurring in cancer is described in the chapter on that disease.

Severe hemorrhages, often so profuse as to be fatal, may occur from pore-like erosions of the mucosa, often so minute as almost to baffle detection. These cases, described by Dieulafoy under the name of *exulceratio simplex*, are described under a separate heading (p. 105). Under the same heading are also included the general oozing of blood from the gastric mucous membrane to which attention has been called by Hale White,¹ and designated by him *gastrostaxis*. In the same category are placed the various post hemorrhages of gastric origin that follow abdominal operations, and the *hematemesis* that not infrequently complicates the course of chronic appendicitis. For a consideration of the above cases, the reader is referred to the section on Hemorrhagic Erosions (p. 192).

Hemorrhages from the stomach, not due to ulcer, cancer, or pore-like erosions, may occur from rupture of venous varices in the stomach itself, from rupture of aneurysm of any of the neighboring arteries into its lumen, or from hemorrhagic blood diseases, such as hemophilia, the various forms of purpura, splenic anemia, leukemia, and intense infections accompanied by extreme hemolysis.

Occult Hemorrhages.—Occult hemorrhages are much more frequent than those that are evident and visible, and their detection constitutes one of the greatest advances made in gastric clinical work.

It is a well-known fact that blood may be present in the bowel movements and yet be undetected, as owing to alterations in its color, and to the small amount present, no evident changes are observed in the color or appearance of the stools, and it is only by delicate chemical tests that its presence becomes known.

The examination for occult hemorrhages in gastric ulcer must, of course, be preceded by an exclusion of all other sources for blood, such as bleeding gums, epistaxis, traumatism, such as the passage of a stomach-tube, intestinal ulcers, hemorrhoids, and rectal fissures. Systemic diseases such as purpura, typhoid fever, arteriosclerosis, and tabes with gastric crises must also be considered. The ingestion of raw or uncooked meat, beef juice, or sausage will give positive reactions.

Thoroughly cooked meats do not interfere with the test, but in doubtful cases in which much depends upon the result of the examination it is best to run no chances and to exclude all meats from the diet for two days preceding the examination. This naturally need not be done unless positive blood reactions are present on a mixed diet.

¹ *Lancet*, 1906, No. 3.

For the examination to be of any clinical significance the following precautions should be taken.

1. No stool should be examined which shows any trace of blood on its surface.

2. Always test from the centre of the stool cylinder.

3. If the patient be at all constipated, give a mild laxative, just sufficient to soften the movement, so that it does not abrade the lower portion of the bowels and cause bleeding.

Tests.—The tests ordinarily employed are benzidin, the aloin, and the guaiac tests. The spectroscopic test and the microchemical test of Teichmann are not as frequently used as formerly and need not, therefore, be described.

Benzidin Test.—Knife point of pure benzidin (Merck) is dissolved by gentle shaking in 2 or 3 cm. of glacial acetic acid. A small piece of well-mixed feces is then ground with distilled water to the consistency of a thin paste, placed in a test-tube, and boiled for about a minute. The contents are then diluted with an equal quantity of water and cooled. 1 c.c. of the benzidin solution is poured in a clean test-tube, 3 to 10 drops of the feces solution added, and after they have been well shaken, 1 to 3 c.c. of a 3 per cent. solution of hydrogen peroxide are added, and the whole well shaken.

In the presence of blood, a green, blue-green, or dark blue color appears, the blue being more marked the larger the amount of blood present. Large quantities of blood show an almost instantaneous reaction—the very small quantities give a decided reaction within two minutes. A negative test gives no blue or green color, which alone is characteristic, even on standing twenty-four hours.

The quantities of the reagents must be carefully observed and the test-tubes and apparatus absolutely clean. Test-tubes in which Fehling's solution has been boiled should not be used.

The following substances besides blood which give the reaction are oxidizing ferments (these are destroyed by boiling), potassium iodide, animal charcoal, iron and copper salts. Potato and farina are said to give a slightly positive test. Pus, saliva, and nasal secretions may give a more or less marked reaction.

The benzidin test gives a reaction of 1 to 200,000 of blood. The only objection to this test is that it is somewhat overdilicate for ordinary clinical work.

Aloin Test.—About 5 grams of the feces are ground up with distilled water until they are of a semifluid consistency, and poured into a large test-tube. The fat is then removed by shaking with an equal volume of ether, and after standing the ether is poured off. The residue is then mixed with one-third of its volume of glacial acetic acid and well

shaken. The aloin solution is prepared by dissolving a knife point of Barbadoes aloes in 5 c.c. of 70 per cent. alcohol. This must always be freshly made. The acetic acid-ether extract is then poured off the feces and 2 or 3 cm. of old ozonated turpentine is added. In the presence of blood a pink, deepening to cherry-red color appears, usually within a few seconds, although in less marked cases the color reaction may not appear in less than five to ten minutes. If a reaction does not occur within fifteen minutes, it should be declared negative. The aloin test gives a reaction of 1 to 25,000 of blood, which is sufficiently delicate for ordinary clinical work.

Guaiac Test.—The guaiac test is performed as the aloin test except that a fresh solution of guaiac in alcohol is employed instead of the aloin. In the presence of blood a blue color appears, although in weak reactions the blue may be masked by the stercobilin of the feces, and is then seen as an olive green or a greenish purple.

The guaiac test is of the same sensitiveness as the aloin test.

Benzidin Paper Test.—Benzidin paper is made by moistening filter paper with a saturated solution of benzidin in glacial acetic acid. During the process contact with the fingers must be avoided, as perspiration gives a positive reaction. A piece of the dried benzidin paper is immersed in the fecal solution to be examined, and then a few drops of a 3 per cent. solution of hydrogen peroxide are added. A positive reaction should appear within one minute—otherwise the test should be considered negative. In doubtful reaction controls by one of the other tests should be employed.

Results of the tests for occult bleeding in ulcer are to be regarded as negative only after three or four tests at least are taken, as experience has shown that positive reactions occur more intermittently in ulcer than in cancer. The tests are valuable not only as an aid to diagnosis, but as an indication as to whether during treatment the ulcer is healing or is still open. Rutimeyer found a positive reaction in 42 per cent. of his ulcer cases.

During the course of an ulcer treatment the stools should be repeatedly examined for occult bleeding, especially after a return to solid diet. If the hemorrhage returns on a solid diet, it should disappear again on resumption of liquid food. Suspicion of malignancy should be entertained toward those cases of presumed ulcers which bleed when solid food is resumed after an ulcer cure. *If an ulcer case of adult years does not improve on an ulcer treatment, but while on meat-free diet shows occult hemorrhages, the case should be looked upon with suspicion, and should bring up for consideration the question of exploration.* In this way many cases of carcinoma will be discovered in their operative period.

GASTRIC ANALYSIS

The passage of a tube during acute ulceration of the stomach is a hazardous procedure and absolutely unjustifiable on any grounds whatever. Unfortunately a large number of casualties have followed the disregard of this rule. Equally dangerous is it to pass a tube during the acute exacerbations of chronic ulcer in which an increase in the severity and constancy of the pain and in localized epigastric tenderness are warning signs of possible perforation. The tube should, moreover, never be passed in cases with recent hemorrhage.

In chronic ulcer the passage of a tube is not especially dangerous, and the writer has never seen any unpleasant results follow its use. Upon the least evidence of pain or hemorrhage the tube should at once be withdrawn.

Gastric analysis may or may not be of service in diagnosis. Valuable assistance may be afforded in determining the presence of an ulcer and its probable location, but it should be remembered that normal gastric secretions offer in themselves no reason for doubting the existence of an ulcer whose presence is suggested by the clinical history.

Hyperacidity.—There has been a great deal written on the relation of hyperacidity to ulcer, the consensus of opinion being that hyperacidity is the rule. It has been argued that hyperacid conditions, howsoever induced, precede the formation of ulcer, and that the hyperpeptic gastric juice with its increased eroding quality is an important factor in its genesis. This is doubtful. In atonic conditions and in gastropotosis, in which hyperacidity is so frequent and in other cases of functional hyperacidity there seems to be no more liability to ulcer than when the gastric juice is normally acid, neither is it necessary to have a hyperpeptic secretion for the erosion of a part of the gastric mucosa because, if an area of diminished local vitality is present, gastric juice of normal strength will do its eroding work quite as well.

There is no doubt that the majority of chronic ulcers are accompanied at some period or another in their course, by increased acidity of the gastric secretions, but this hyperacidity is not constant, nor is it ordinarily observed in the acute forms of ulcer, while not infrequently ulceration takes place when the acidity of the gastric juice is greatly diminished.

Elsner, Strauss, and Rüttimeyer, writing independently of each other, have called attention to the differences in this regard in ulcers from various geographical regions, higher acidities being more common in England and the United States, apparently, than in Germany.

The proportion of ulcers accompanied by hyperacidity is not an

overwhelming one, the estimates varying from 35 to 75 per cent. The principal statistics are shown in the following table:

Ewald found hyperacidity in	34 per cent.
Moynihan ¹ found hyperacidity in	40 per cent.
Wagner found hyperacidity in	42 per cent.
Wirsung found hyperacidity in	42 per cent.
Rütimeyer found hyperacidity in	42 per cent.
Oerum found hyperacidity in	56 per cent.
Fenwick found hyperacidity in	66 per cent.

In the writer's series, hyperacidity of 70 or over was found in 32 per cent. of the acute ulcers, and in 50 per cent. of the chronic cases. The author's statistics are as follows:

ACIDITY OF TEST BREAKFAST IN ACUTE ULCER

Total acidity	40 to 50	17 per cent.	} 68 per cent.
Total acidity	50 to 60	34 per cent.	
Total acidity	60 to 70	17 per cent.	
Total acidity	70 to 80	16 per cent.	} 32 per cent.
Total acidity	80 to 90	9 per cent.	
Total acidity	90 to 100	7 per cent.	
		100 per cent.	100 per cent.

ACIDITY OF TEST BREAKFAST IN CHRONIC ULCER

Total acidity	30 to 40	10 per cent.	} 50 per cent.
Total acidity	40 to 50	14 per cent.	
Total acidity	50 to 60	14 per cent.	
Total acidity	60 to 70	12 per cent.	} 50 per cent.
Total acidity	70 to 80	16 per cent.	
Total acidity	80 to 90	10 per cent.	
Total acidity	90 to 100	8 per cent.	} 50 per cent.
Total acidity	100 to 110	8 per cent.	
Total acidity	110 to 120	8 per cent.	
		100 per cent.	100 per cent.

In these tables are included all cases of ulcer of stomach or duodenum, irrespective of their situation as regards the pylorus. It will thus be seen that in acute ulcer the acidity is not as high as in the chronic form, and acidities over 100 did not occur.

After a careful analysis of his cases, the writer believes that hyperacidity is not a necessary result of ulcer. When an ulcer is situated in a location which does not involve the pylorus, hyperacidity is rather rare, but whenever an ulcer of the stomach infringes upon the pylorus

¹ Lancet, January 6, 1912.

so as to interfere in the slightest degree with its patency, or whenever by its propinquity it causes pyloric spasm, a definite hyperacidity results.

Hyperacidity in ulcer depends upon the *localization of the ulcer* rather than upon the ulcer *per se*.

The writer's conclusions are based upon the following statistics:

ULCERS NOT INVOLVING THE PYLORUS

Low acidity	9 per cent.
Normal acidity	75 per cent.
Hyperacidity	16 per cent.

ULCERS AT OR NEAR THE PYLORUS

Low acidity	0 per cent.
Normal acidity	18 per cent.
Hyperacidity	82 per cent.

The author believes that the importance of the location of the ulcer influencing hyperacidity has not been as yet fully recognized. It is acting upon this hypothesis that he is led to describe the results of gastric analysis in ulcer under two separate headings.

1. In ulcers, gastric or duodenal, that are not situated near the pylorus, and which therefore do not directly or indirectly impair the patency of that orifice, gastric analysis is of very little service whatever. The fasting stomach is empty. The test breakfast presents a normal appearance, and is usually of normal acidity. An increase in gastric mucus is not noted. The only help in diagnosis that may be afforded is the presence of old altered blood in the gastric contents.

2. Of more importance are the gastric tests made with ulcers, gastric or duodenal, that directly or indirectly affect the patency of the pyloric orifice, either by thickening of the tissues of the pyloric ring, cicatricial contraction, vascular tumefaction, or spasm. Hyperacidity is the rule. Hypersecretion occurs in a large number of the cases.

The fasting stomach may be empty, or may contain from 25 to 50 c.c. of a clear fluid of normal or excessive acidity and without gross admixture of food residue, although a few scanty food remains may be found under the microscope. This mild hypersecretion occurred in 50 per cent. of the total number of the author's chronic ulcer cases. Presence of residual food implies more advanced pyloric stenosis.

This slight excess of gastric secretion in the fasting stomach, associated clinically with hunger pain, affords a strong presumptive proof of ulcer provided we can exclude chronic inflammation of the appendix.

Test breakfast is usually well digested and free from gastric mucus. It may be of normal appearance, but more usually it separates upon

standing into two layers, one of fluid above, and one of digested breadstuff below. A certain degree of alimentary hypersecretion is common to all atonic conditions of the stomach, but in atony the depth of the supernatant layer should never exceed that of the lower. In ulcer the amount of hypersecretion may be more than this, the depth of the liquid layer being occasionally four to twenty times that of the underlying stratum. These excessive amounts of alimentary hypersecretion are not common, but they do occur.

The acidity of the filtrate is usually above the normal, total acidities of 80 to 95 being the rule. More rarely higher figures are attained, 100 to 120.

Whenever spasm of the pylorus or tumefaction of the orifice occurs, the acidity of the test breakfast or of the vomited matters becomes excessively high, occasionally attaining 130 to 150, and the increased acidity is often complicated by an increase in the actual quantity of the gastric juice that is poured out. As conditions improve, hypersecretion diminishes.

The following case may be quoted as an example. W. E. C. C., aged forty-five years. Five years ago suffered for one month with constant gnawing pain in the stomach before meals, relieved by the taking of food. Four years ago this pain reappeared and has remained ever since with but short periods of intermission. Two years ago he had a large intestinal hemorrhage.

Fasting stomach empty.

Test breakfast, 100 c.c., well digested, separates on standing into layers of equal depth. No mucus. Total acidity, 48; free hydrochloric acid, 24. Blood test negative.

Was placed on ulcer cure. On the third day of his starvation, vomited 14 ounces of a clear greenish fluid (total acidity, 98), followed in a few hours by the vomiting of 2 pints of similar fluid of the total acidity of 132. Under appropriate treatment the vomiting ceased, and for the past seven years there has been no recurrence. Gastric analysis at the end of a month's ulcer cure showed total acidity, 78; free hydrochloric acid, 48.

A differential diagnosis between gastric and duodenal ulcer cannot be made by examination of either the fasting or the digesting stomach. Inasmuch as the majority of duodenal ulcers are situated near the pylorus, tumefaction or spasm is apt to occur, diminishing the lumen of that orifice and allowing of a higher acidity than is found in gastric ulcer taken as a whole, although between duodenal ulcer and gastric ulcer equally near the pylorus there seems to be no difference in acidity.

Neither is it possible to differentiate with certainty between ulcer and cancer of the stomach by gastric analysis alone. The writer has

found in cancer that 15 per cent. of the test breakfasts show nothing that is indicative of malignancy; hydrochloric acid is present; lactic acid is negative; bacilli and evidences of stagnation are not present. In the remaining 85 per cent., however, more positive evidences of malignancy are found.

In chronic ulcer it is important to follow our cases, and to make gastric analyses from time to time. It has been stated that whenever malignancy develops on the site of an old ulcer, the first change evident is a gradual, though steady reduction in the acidity of the gastric secretions.

The writer cannot give his personal experience on this point, as he has seldom, if ever, noticed such a primary lowering of acidity in these cases. In his experience, however, the first evidence of beginning malignancy has been traces of lactic acid. Lactic acid, even in small traces in the stomach contents of ulcer patients, should always suggest malignancy.

PHYSICAL SIGNS OF ULCER

Physical evidences of gastric or duodenal ulcer are so frequently lacking that their absence should never exclude the possibility of an ulcer lesion being present. Tenderness and other physical signs, when elicited, afford only confirmatory proof.

The most typical physical sign is localized tenderness. This is usually found over a small area just below the xiphoid, or midway between the xiphoid and the navel, corresponding to the position of the celiac ganglion. In duodenal ulcers the tenderness extends somewhat to the right, although for unexplained reasons tenderness in duodenal ulcers may be confined entirely to the left hypochondrium. Care should be taken not to confuse this local tenderness with the more diffuse tenderness that is elicited by the deep palpation of the abdominal aorta in thin nervous women or with the smaller area of localized tenderness in the median line due to small epigastric hernia.

The area of tenderness affords no clue whatever to the localization of the ulcer.

More rarely the whole epigastrium is diffusely tender—a sign which is devoid of any diagnostic value whatever.

Associated with the tenderness there may be a slight amount of localized rigidity of the abdominal wall. The association in duodenal ulcer, of a tender area 2 to 3 inches in diameter in the median line and to the right just above the navel, with slight rigidity of the upper portion of the right rectus muscle, and a heightened epigastric reflex on that side, is not an uncommon one, but these signs are often lacking, or present only during the period of greatest pain. Tenderness is

usually most marked in acute ulcers or during the acute exacerbations of the chronic form. In these acute stages of ulceration there may be found a zone of cutaneous hyperesthesia 2 to 3 inches in breadth, starting in front at the median line in the epigastrium, and following the course of the intercostal nerves to the back, terminating in an intensified area of sensitiveness at the point of emergence of the posterior nerve filaments. The boundaries of this zone, described by Head, are determined by gently scratching the skin with a pin in vertical lines from above downward and then from below upward, and marking the points above and below at which the sensation of ordinary scratching passes into one of greater intensity of pain, often likened by the patient to the rubbing of an abraded surface. This area of hyperesthesia is not distinctive of ulcer, as it may be present in appendicular dyspepsia.

A dorsal point of tenderness first described by Boas may be found at the level of the eleventh and twelfth dorsal vertebrae a little to the left of the spinal column, having a lateral expansion of 2 or 3 cm. and a height of 1 to 4 cm. It may be found on both sides, and in some cases, especially in duodenal ulcer, according to Elsner, the dorsal point may be present only to the right of the median line. According to Boas it is present in one-third of all cases of ulcer, although in the writer's experience it is found with but half this frequency. It is generally more common with ulcers of the posterior wall, and if limited entirely to the back, it may be of the greatest diagnostic importance.

If Head's hyperesthetic zone is present the dorsal point marks the posterior limit of the hypersensitive area.

In acute ulcer there is no tumor mass to be felt, but such is not the case in many instances of chronic ulcer. Callous ulcers with thickened bases may be palpable through the abdominal wall, as may those ulcers which have contracted adhesions with neighboring parts. The tumor that can be thus palpated may so closely resemble malignancy that a differential diagnosis is impossible—in fact an exact diagnosis may only be determined by microscopical examination. In the cases that have come to operation the error has invariably been in one direction, that is to say, a non-malignant mass was believed to be cancerous.

The physical signs of the complications will be described under their separate headings.

RADIOGRAPHY OF ULCER

Were it a fact that the roughened base of an ulcer regularly retains its bismuth coating after the rest of the meal had left the stomach,

the radiographic diagnosis could be easily and definitely established. Unfortunately the ordinary crateriform ulcer does not retain its bismuth coating for any longer time than does the mucous membrane in its vicinity, so that we are not furnished with any definite signs that it exists. It is only by the indirect evidences of ulceration that we may be suspicious of its presence. These signs are not diagnostic in themselves, so that a decision cannot be reached by the study of the plates alone; it is only by the combined evidence of clinical history, physical examination, gastric analyses, and the radiographic plates that correct conclusions can be drawn. The physician has no right to expect a diagnosis to be made for him by a radiologist who knows nothing more of the patient than can be learned by the study of the plates. The physician and radiologist must work in conjunction with each other, and each supplement by his special knowledge, the acquirements of the other.

Radiological Diagnosis of the Stomach.—Technique.—Following the teachings of Holz knecht and Haudek, the author recommends the following as a routine technique in all stomach examinations. The patient is first prepared by a thorough catharsis, preferably by castor oil given at night. The following morning at a prearranged hour the patient takes a Rieder meal of 8 ounces of oatmeal gruel into which is thoroughly mixed 2 ounces of bismuth subcarbonate or bismuth oxychloride, obtained from a reputable druggist, so that the drug is as pure as can be obtained. A light breakfast of tea and toast may be given one hour later. The patient is to be at the radiologist's office five and one-half hours after taking the Rieder meal, so that the first radiograph may be taken exactly six hours after the ingestion of the bismuth. This plate will show the motility of the stomach and the location of the head of the bismuth column in the ileum or colon. A second bismuth meal, composed of bismuth subcarbonate or oxychloride $1\frac{1}{2}$ ounces, gum acacia mucilage 2 ounces (= 33 per cent. gum acacia), water q. s. to 8 ounces, is then given, and a second radiograph immediately made, which in its turn will show the size, shape, and position of the stomach. There are now in the two plates, as a rule, sufficient radiological data, combined with the history, clinical findings, and appearance of the patient, to make a diagnosis of the case. Occasionally a third radiograph may be taken fifteen minutes after the second as a control, or to see the motility of pylorus and first part of the duodenum. Sometimes in cases of hypermotility it is well to radiograph the patient three hours after the ingestion of bismuth. The patient is radiographed standing, although additional plates may be taken in the recumbent position if desired.

· **Radiographic Indications of Ulcer.**—The following radiographic findings may be considered suspicious of ulcer:

1. Bismuth residue in the stomach six hours after the meal indicates a lack of motility which may be due to spasm, tumefaction, or slight cicatricial contracture of the pylorus, or to atony. If the stomach show a normal outline, atony may be excluded.

Unfortunately there are some instances of pylorospasm secondary to chronic appendicitis or irritative lesions of the gall-bladder, in which bismuth remains may be found in the stomach six hours after the meal, and in which the contour of the stomach is normal, so that by the *x*-ray alone a differential diagnosis cannot be made. Holzknacht lays stress, however, upon the presence of tenderness on palpation on a spot which radiographically corresponds to the lesser curvature near the pylorus, and which shifts its position according to the varying positions of the stomach. It is of the greatest service carefully to palpate the epigastrium before the *x*-ray examination, and to mark the point of maximum tenderness by a small bird-shot applied to the spot by an adhesive strip.

2. A displacement of the pylorus upward and to the left is not infrequent with ulcers of the lesser curvature, which cause contraction along the upper border approximating the cardia and the pyloric end. The resulting shape of the stomach is sometimes spoken of as the "snail form." The last portion of the greater curvature, instead of curving upward to the right, will be drawn perpendicularly upward and to the left, giving that portion of the stomach the undershot appearance of a bull-dog's jaw. This undershot appearance of the greater curvature is also seen with pyloric obstruction.

3. Hour-glass contraction that appears in all of a series of plates are suggestive of old cicatrizing ulcer.

A spastic hour-glass contraction is frequently seen with ulcer of the lesser curvature, especially if adherent to the under surface of the liver. There is a contraction or drawing in of the greater curvature, as if the lower border were pulled up at this point, allowing of sagging on either side. It is to be distinguished from a peristaltic wave by the fact that the indentation appears on the greater curvature only. Spasmodic hour-glass comes and goes, is present on some plates and not on others, and will remain unchanged whether the patient stands or lies down. The same incisure may also occur on the lower curvature from spasm originating from ulcer of the lesser curvature, even in the absence of adhesions, and this may appear fixed in a large number of plates. A similar incisure may also appear in the plates of gastric cancer.

PLATE I

Fig. 1

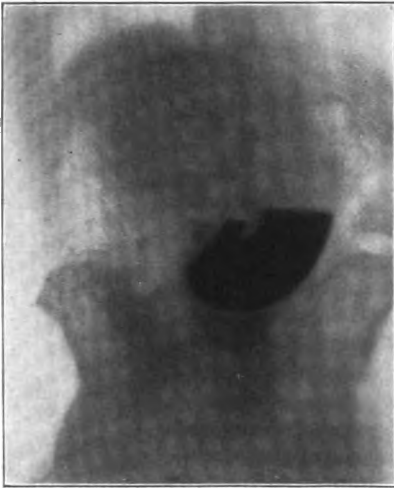


Fig. 2

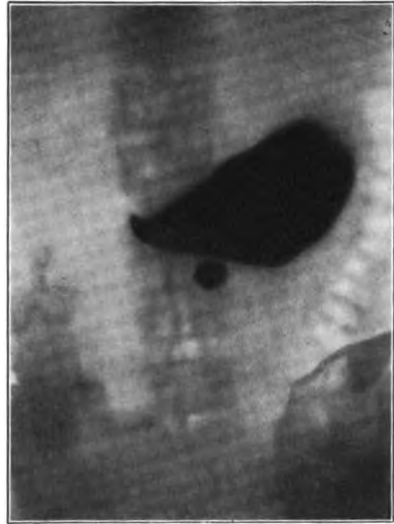


Fig. 1.—Ulcer of Lesser Curvature, Retraction of Lesser Curvature, drawing Pylorus up and to the left. The so-called "snail-form," described by Haudek. (Radiologist, Dr. Leaming.)

Fig. 2.—Ulcer of Lesser Curvature and Pylorus. The typical bowl-shaped bismuth residue is not as marked as is usual, although pyloric stenosis exists. (Radiologist, Dr. Busby.)

Fig. 3

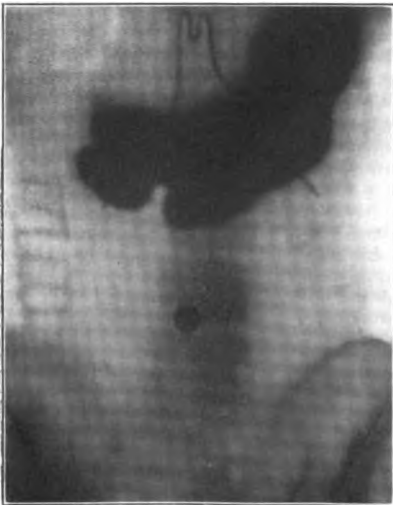


Fig. 4



Fig. 3.—Ulcer of Lesser Curvature. Spasmodic Incisure of Greater Curvature. (Radiologist, Dr. Busby.)

Fig. 4.—Ulcer of Pylorus with Adhesions and Distortion, Closely Resembling Carcinoma. (Radiologist, Dr. Busby.)

Hertz¹ has shown that an ulcer of the lesser curvature adherent to the liver may, when the patient stands, produce a line of tension diagonally downward, or up the greater curvature at this point, while on either side a sagging of the lower border may appear, causing the semblance of an hour-glass stomach, which disappears when the patient lies down.

4. Distortion or displacement of the stomach by adhesions suggests ulcer as a probable cause.

In the majority of cases of adhesions following ulcer, the region of the cap and pyloric sphincter are first involved. The cap looks contracted, its edges are ragged, or its surface worm-eaten, showing different degrees of density. The sphincter may appear rough and indistinct in outline and the lumen may be displaced from its central position.

The lesser curvature of the pyloric portion is more likely than the greater curvature to be involved, so that the indistinct area of the sphincter may be wedge-shaped, the broader base being the uppermost.

Frequently the contour of the stomach will reveal the identity of the adherent viscus.

Adhesions to the gall-bladder or liver generally result in the stomach lying horizontally, rather than in the normal oblique or vertical position. This abnormality must, however, be present in all plates before any inference can be drawn.

Adhesions from gall-bladder infection may give the same radiographic findings as those due to ulcer, although in many instances suspicion is aroused by the angulation of the cap to the right, so that the lumen runs horizontally instead of vertically. The cap may be contracted and irregular, becoming, as the adhesions progress, quite asymmetrical and ill-defined.

Gallstones are only infrequently revealed by the x-ray, but occasionally calcareous deposits form, so that they may be recognized and throw considerable light upon the diagnosis.

Adhesions are often progressive, so that successive examinations should be made at intervals.

The diagnosis of adhesions by x-ray is not, however, quite so simple as it would appear, and many adhesions are supposed to exist in cases in which normal conditions are found at exploration. A stomach may appear as if fixed in an abnormal position and still may be proved to be freely movable by changes in the position of the patient. The Trendelenburg position is often serviceable in demonstrating the mobility of a stomach that at first may seem adherent.

The diagnosis of adhesions cannot be made on the evidence of one

¹ Lancet, April 6, 1912.

plate alone. Successive examinations must be made and the findings must be constant.

5. A small puckered area in which the rugæ are distorted is suggestive of gastric ulcer. If the area be large, the region may fail to expand and contract with the rest of the organ. It is even more suggestive when the localization of the affected area coincides with that of local tenderness on palpation.

6. Haudek makes a point of watching for an upper line of fluid across the stomach after a liquid bismuth meal, thus indicating hypersecretion. This test is not a very conclusive one, and does not compare with the simpler method of testing for hypersecretion by the passage of a tube in the fasting state.

7. Fluoroscopy may show at times a reversed peristalsis, indicating an extreme degree of spasticity. This is suggestive but not conclusive.

8. The radiographic findings of ulcer involving the patency of the pyloric canal resulting in stenosis, will be described under the latter heading.

Radiographic Examination of Duodenal Ulcer.—1. One of the most characteristic appearances is an upward displacement of the pyloric end of the stomach and its retention by adhesions, so that the stomach lies obliquely, or even horizontally, rather than vertically. The importance of this finding is generally acceded.

2. Fluoroscopy may demonstrate intermittent pyloric contraction. The stomach may start to empty itself, when suddenly pylorospasm occurs, preventing further egress, until after a variable time the spasm relaxes, and the bismuth is again forced into the duodenum.

3. An indentation in the cap caused by the descent at one side of the second portion of the duodenum should not be confused with a pathological condition.

4. A shadow of bismuth visible in the cap five or six hours after the ingestion of the bismuth meal when the stomach and the remainder of the duodenum are completely evacuated, has been said to indicate the presence of a duodenal ulcer.

5. Haudek has seen with the fluoroscope, sharp contractions of the duodenum in cases of ulcer, but considers the phenomenon exceedingly rare.

6. Radiographic evidence of hypersecretion should suggest the possibility of duodenal ulcer.

Radiographic Diagnosis of Penetrating or Perforating Ulcer.—When an ulcer has extended through the wall of the stomach and opens into a cavity limited by adhesions, or perforates into the liver or pancreas, definite and convincing evidence may be obtained by the x-ray. Six hours after the ingestion of the first bismuth meal there

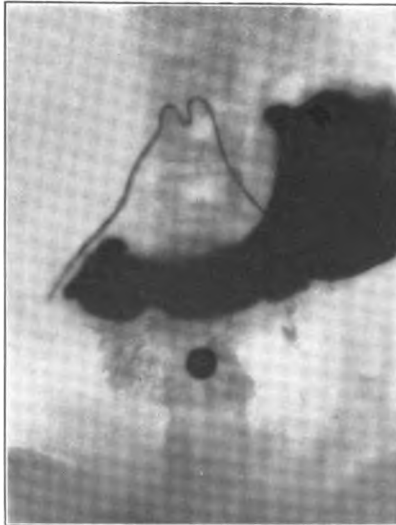
PLATE II

Fig. 1



Duodenal Ulcer, after Perforation. Stomach distorted and misshapen by adhesions. (Radiologist, Dr. Le Wald.)

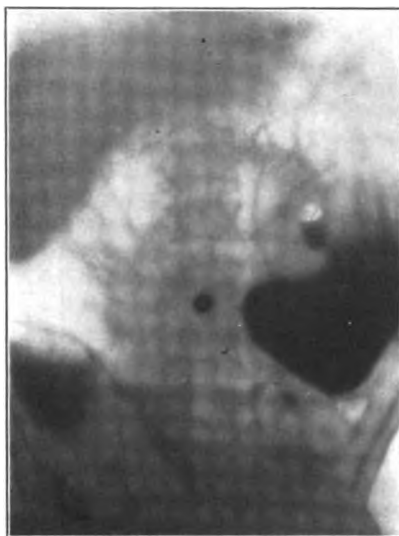
Fig. 2



Ulcer of the Duodenum, showing Transversely Lying Stomach, with Duodenum far to the Right of the Type Described by Holzkecht. (Radiologist, Dr. Busby.)

PLATE III

Fig. 1



Ulcer of Lesser Curvature Penetrating into the Liver. Noteworthy is the isolated bismuth residue above the line of lesser curvature, surmounted by an air-bubble. (Operator, Dr. George E. Brewer; radiologist, Dr. Leaming.)

Fig. 2



Fig. 8

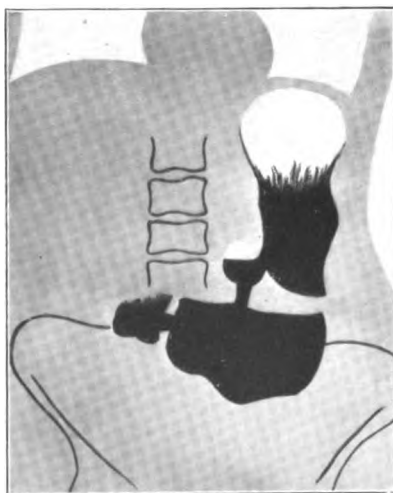


Fig. 2.—Six-hour Plate of Ulcer of the Duodenum Penetrating into the Pancreas. Noteworthy are the bismuth residue in the stomach and the isolated bismuth residue in the false cavity surmounted by an air-bubble. (Operator, Dr. Charles H. Peck; radiologist, Dr. Leaming.)

Fig. 8.—Penetrating Ulcer, with Large Cavity in the Liver. Hour-glass Contraction. Pylorus Free. Bismuth Residue after Six Hours. (Drawn from Holzknecht's article, Archives of the Roentgen Ray, April, 1912, p. 67.)

may be seen a small isolated patch of bismuth, usually of a half-moon shape, often surmounted by an air bubble, which persists after the complete evacuation of the stomach. When the examination is made immediately after the filling of the stomach with bismuth suspension fluid, the patch is often seen just outside of the contour of the stomach wall. These findings are well illustrated in the accompanying plates. Such a diagnosis of perforative ulcer by x-rays, according to Holzknecht,¹ is not unusual, for he reports that Haudek has met with 28 cases of this variety, the diagnosis being confirmed in 15 instances. In many of these cases there was an absence of hydrochloric acid in the gastric juice.

PROGNOSIS AND END-RESULTS

Prognosis.—The prognosis of gastric and duodenal ulceration both as regards the percentage of mortality and of the curative effect of treatment is extremely difficult to determine. A mass of statistics is available, but in many respects they are often both insufficient and misleading. This applies both to the reports of medical and of surgical cases.

Immediate Prognosis under Medical Treatment.—Medical statistics are especially lacking in accuracy for the following reasons:

1. When a surgeon operates upon a patient, he sees an ulcer and recognizes it as such. The physician, on the other hand, who makes his diagnosis without the benefit of such palpable and visible evidence, may or may not be correct in his conclusions. Many cases are treated as ulcers that are really suffering from chronic appendicitis or gall-bladder disease. A few years ago the writer, wishing to trace the end-results of ulcers treated by him, wrote a series of letters to those cases only in which the diagnosis seemed certain. 12 per cent. of replies were to the effect that after the ulcer cure the symptoms had improved but had not ceased until, after an attack of appendicitis, the removal of the appendix had been followed by an entire and permanent cessation of gastric symptoms. This bitter experience has never been forgotten.

While this inclusion of cases resembling ulcer but incorrectly diagnosed does not add to the percentage of mortality, but probably rather to the apparent reduction of the death rate, nevertheless it is obvious that it materially reduces the number of cases that are neither improved nor cured by the ulcer treatment.

12 to 15 per cent. of all cases diagnosticated as ulcer give disappointing end-results because of this error in diagnosis.

¹ Archives of Röntgen Rays, July, 1912.

2. The prognosis is different according to whether the figures are compiled from private or from hospital sources.

In hospital cases the mortality is naturally greater, because of the greater severity of the cases admitted and of the larger proportion of cases entering with severe hemorrhage or perforation. Of 100 consecutive cases of ulcer admitted to Bellevue Hospital and compiled by the writer, 17 per cent. were admitted with hemorrhage and 7 per cent. with perforation.

The end-results of treatment are very different in the two classes. For medical treatment to effect results in chronic ulcer, it should be continued for a long period of time, even after the cessation of actual symptoms. Hospital cases remain in the wards just as long as they suffer pain—and leave as soon as they are fairly comfortable, returning to a promiscuous, unsuitable diet, and a disregard of all medical conventions. No wonder so many are unimproved and no wonder so many relapses occur. It is only after alarming hemorrhage that many of the hospital patients will undergo any treatment at all.

The comparatively small number of perforations which occur in patients who have had ulcer treatment because of hematemesis shows the beneficial effects of medical treatment, even if it be inadequate to effect a permanent cure.

The differences between hospital and private cases are well shown by the following tables arranged by Musser:

SIMPLE ULCER WITHOUT COMPLICATIONS, FROM GENERAL SOURCES—409 CASES

	Cured. Per cent.	Improved. Per cent.	Unimproved. Per cent.	Died. Per cent.
Treated medically	73.3	7.9	6.4	12.4
Treated surgically	68.1	5.1	6.6	20.0

The total mortality of medical and surgical cases was 17.3 per cent.

SIMILAR ULCERS TREATED IN PRIVATE PRACTICE—194 CASES

	Cured. Per cent.	Improved. Per cent.	Unimproved. Per cent.	Died. Per cent.
Treated medically	60.1	32.0	5.0	3.1
Treated surgically	77.1	8.5	2.9	11.4

For a very complete and interesting series of statistics the reader is referred to Musser's article.¹

3. It is recognized that simple acute ulcers usually heal readily under medical treatment, and that in these cases the prognosis, as a rule, is good. Chronic ulcers, with or without complications such as

¹ Amer. Jour. Med. Sci., December, 1907, cxxxv, 781.

pyloric narrowing, hour-glass contractions, and extensive adhesions, are more intractable and frequently require operative interference. To bring these two classes of cases together in one compilation, naturally gives results that are erroneous and misleading. It is not surprising, therefore, that the death rate should be so variously estimated, the percentages running in a long series from 1 to 50 per cent. It is, however, generally conceded that while in hospitals the death rate may be as high as 20 per cent., according to the class of patient admitted, the average percentage of mortality is not far from 10 per cent. Musser, from a large compilation of statistics, estimates that about 8 per cent. of ulcer patients die as a result of the disease.

When the medical treatment can be carried out the results in individual hands may be somewhat less. Von Leube, whose skill in the treatment of ulcer is well recognized, lost but 2.4 per cent. of a total number of 556 cases treated by him. In the writer's experience, the mortality of ulcers seen in private practice amounted to 3.1 per cent. In hospital practice, excluding those cases in which the cause for death was an intercurrent disease not associated in any way with chronic ulceration (3 cases of acute lobar pneumonia, 1 of chronic nephritis and uremia, and 1 from arterial sclerosis and senility, in all of which cases a chronic ulcer was found which had not given symptoms during life) the ulcer caused death in 11.1 per cent. The general mortality of both private and hospital cases amounted to a little less than 7 per cent. These figures are identical with those of Musser.

ESTIMATES OF MORTALITY IN ULCER MEDICALLY TREATED

	Per cent.
Hewes	2.0
Robinson	2.1
Russell	2.1
Von Leube (556 cases)	2.4
Musser, private sources	3.1
Lockwood, private sources	3.1
Schultz, Breslau, and New Hamburg Hospitals	5.4
Greenough and Joslin, Massachusetts General Hospital	8.0
Lockwood, hospital cases	11.1
Musser, hospital cases	12.4
Hawkins, St. Thomas' Hospital	13.3
Welch	15.0
Sears, Boston City Hospital	21.0
Thompson	20.5
Robson (estimated)	25.0
Musser, both hospital and private cases	8.0
Lockwood, both hospital and private cases	7.0
Average mortality in both hospital and private cases generally conceded	8 to 10.0

Immediate Prognosis under Surgical Treatment.—The results of operation for gastric ulcer are far better in the case of a few experienced operators than the larger number of operations performed by the surgical profession at large.

The figures of some surgeons show a remarkably low rate of mortality. The following table is given by Deaver:

STATISTICS OF OPERATIONS FOR BENIGN DISEASES OF THE STOMACH

Operator.	Date.	Number of operations.	Deaths.	Mortality. Per cent.
Crile	1908	56	1	1.7
Czerny	1902	83	4	4.8
Deaver	1900-1907	91	8	8.7
Hartmann	1903-1905	47	3	6.3
Helferich	1905	86	7	8.1
Hochenegg	1906	94	6	6.4
Krause	1906	55	5	9.0
Mayo	1906	307	19	6.2
Morison	1905	27	1	3.7
Moynihan	1906	334	21	6.2
Power, D'Arcy	1906	41	3	7.3
Robson, Mayo	1906	322	10	3.1
Rotgans	1906	5.0
Schou	1907	54	3	5.5
Schloffer	1906	53	2	3.8

Mayo, in 1906, had a series of 167 gastro-enterostomies with only one death. Mayo Robson reported 2 deaths in 112 gastroduodenostomies for benign disease. Moynihan, in 1906, reported 248 cases, with only 2 deaths, a mortality of only 0.8 per cent. There had been no deaths among the last 151 cases.

In 1000 cases of different types of ulcer treated surgically by W. J. and C. H. Mayo¹ by all varieties of operation, the immediate mortality was 2.4 per cent.

At the same time it must be noted that the mortality of operations done by less experienced surgeons is far greater than this.

French reports from Guy's Hospital a mortality of 23.4 per cent.; in St. Bartholomew's Hospital the deaths after operations were 17.1 per cent.; of 150 benign cases quoted by Bettman and White, the immediate mortality was 10 per cent. Musser's figures of operations done for simple uncomplicated ulcer show that 11.4 per cent. were fatal in private practice, 20 per cent. in hospital cases.

Hall² reports 6 deaths in 50 of his cases operated upon by various surgeons—a mortality rate of 12 per cent.

¹ Annals of Surgery, September, 1911.

² Amer. Jour. Med. Sci., cxxxvii, 625.

A series of figures published by Hartmann show how the danger of operations diminishes with the increasing skill and experience of the operator.

His first series of operations, comprising 21 gastro-enterostomies, with 5 deaths, gave a mortality of 23.7 per cent. His second series consisted of 34 gastro-enterostomies with 3 deaths—a mortality of 8.8 per cent., while his last series included 47 gastro-enterostomies with 3 deaths, showing a mortality of 6.3 per cent.

These figures show how important it is for the physician in charge of an ulcer case requiring surgical interference to choose as his confrère an operator of known skill and experience in gastric surgery.

End-results of Medical and Surgical Treatment.—End-results of Medical Treatment.—It is difficult to arrive at satisfactory conclusions by a study of the statistics available, because, as a rule, the patients have not been followed long enough to determine whether or not the ulcer has actually healed. The relief, or even the total cessation of symptoms, should not bring an erroneous belief that the ulcer is healed and the patient cured, for in many of the patients, even in those who conscientiously carry out the instructions of their physician, symptoms of ulcer sooner or later reappear, and the relapse may be as serious as was the original attack.

How long a time of freedom from symptoms must elapse after treatment before the patient can be declared cured is an arbitrary one, but it may be said that two years at least must elapse without symptoms before any conclusions can be drawn.

It is more difficult to trace hospital patients than those seen in private practice, but when they can be followed, it will be found that relapses are much more frequent than in those whose mode of life and intelligence guard them from the obvious faults of living which militate against those in the lower walks of life. The longer the patients are followed the greater the number of medical failures that become evident. No value can be attached to the reports of cases that are not followed for at least two years. It may be said, from a study of statistics that conform to scientific requirements, that under medical treatment, about 80 per cent. of cases are apparently cured, and that in about one-half of these the symptoms of ulcer again become manifest.

Greenough and Joslin found in the Massachusetts General Hospital that while 82 per cent. of ulcers were discharged as “cured” or “relieved,” but 40 per cent. remained well. Sears estimates the number of medical failures after five years at 50 per cent. Mumford and Howe estimate 80 per cent. of apparent cures, of which one-half relapse. Hewes in 51 cases found 48 apparently cured. In two years 63 per

cent. of these remained well. Paterson in 72 hospital cases discharged cured found that but 19 remained well, 7 were doubtful, 40 were still suffering, 5 had undergone surgical treatment for their complaint, and 1 had died.

In about 20 per cent. the immediate result is not brilliant, a certain amount of distress still remaining, although, as a rule, the patients are benefited to a greater or less extent by their treatment. The number of those who show no improvement whatever is relatively small, probably not over 5 or 6 per cent. It is probable that many of these improved or unimproved cases are not ulcers at all.

In 1897 von Leube, in a communication before the Thirteenth Surgical Congress, announced that whereas the former mortality of ulcer had been as high as 13 per cent., the enforcement of the rigid treatment inaugurated by him had been promptly followed by a marked reduction in the death rate. Of 556 cases reported by him, 74.1 per cent. could be considered cured, 21.9 per cent. improved, 1.6 per cent. unimproved, 2.4 per cent. had died.

In the year 1909 he has given the total of his later results to date,¹ and they show a surprising improvement over previous records.

Of 627 cases both of ulcers with hemorrhage and of those without, treated in the previous eleven years, in his clinic and in his private practice, 566, or 90 per cent., were clinically cured, 76 per cent. within four weeks, 14 per cent. in a period longer than this; 53, or 8.5 per cent., were improved; 6, or 1 per cent., remained unimproved, and 2, or 0.3 per cent., died as the result of hemorrhage.

Tabulating his ulcers that were attended by hemorrhage he found, that 72, or 90 per cent., were clinically cured (66 per cent. within five weeks, 24 per cent. in a longer period of time than this); 5, or 6.25 per cent., improved; 1, or 1.25 per cent., remained unimproved, while 2, or 2.5 per cent., died.

These are astounding results. The writer, however, makes use of the term "clinically cured," because von Leube states in his communication that he regards as "cured" those cases in which the symptoms disappear for a period of three weeks, and in which ordinary hospital diet, not especially prescribed for gastric cases, but given to those whose digestion is good, is eaten without discomfort. He notes that occasionally an ulcer is only apparently cured, and that after a longer or shorter time pain, dyspepsia, and hemorrhage may reappear, but in his experience such a relapse occurs only in very rare exceptions.

It is unfortunate that von Leube has not followed up his cases for two years or more, and given us the end-results thus obtained. The

¹ Deutsch. med. Woch., June 3, 1909, No. 22.

writer's experience leads him to believe that under a rigid ulcer cure fully 90 per cent. of cases may be considered "cured" in the sense in which that term is used by von Leube, but is not so optimistic as to the after-results of treatment.

Of the ulcer cases treated by the writer by a rigid and thorough ulcer cure, and followed for over three years, the following data may be given:

Permanently cured	50.0 per cent.
Temporarily cured but relapsed	16.6 per cent.
Improved	22.2 per cent.
Unimproved	5.5 per cent.
Died	5.7 per cent.

Of the 22.2 per cent. improved and 5.5 per cent. unimproved, a total of 27.7 per cent., it is probable that at least half were suffering from appendicular dyspepsia rather than ulcer, or from a combination of chronic ulcer and chronic appendicitis. Twelve per cent. of the writer's cases of ulcer that did not improve by medical treatment were finally cured by appendectomy. It is reasonable to conclude that many who were not ultimately operated on were nevertheless cases of this description. Unfortunately, only operation or autopsy can decide. The causes for death in the 5.7 per cent. were various and largely due to intercurrent disease, as would be expected, for many of the cases had been traced for ten years or more. The exact data cannot be given, as in many instances the cause for death could not be ascertained. It is a fact, however, that very few, if any, died from stomach disorders, and that cancer did not seem to be the cause for death in any of the cases that could be traced.

The immediate results of treatment were satisfactory in much greater proportions than these. If we should add the end-results of the ulcer treatment as applied to those whose complaint is really chronic appendicitis, or gall-bladder disease, that have been erroneously diagnosticated as ulcer, the proportion of the cases entered on the case books as "improved" would be far greater.

End-results of Surgical Treatment.—Owing to the great advances made in recent times in gastric surgery, and the large number of patients operated upon, only a few can give a postoperative history of sufficiently long duration to be authoritative as to ultimate results. It must be conceded, however, that in no other field of operative work have more brilliant results been effected, both as regards degree of improvement and permanency of cure. Writing, however, under the excitement of their successes, it is possible that too much has been claimed for surgery.

Deaver writes: "Surgery allows 95 to 98 per cent. of patients to recover," and at the same time publishes the following table of surgical end-results, in which the average cure is but 86 per cent.:

END RESULTS OF OPERATIONS FOR BENIGN DISEASES OF THE STOMACH

Operator.	Cases traced.	Cured.	Per cent.
Mayo (1908)	234	189	80.7
Moynihan (1908)	247	211	85.42
Czerny (1902)	53	44	83.0
Robson	96	89	92.7
Deaver (1900-1907)	64	49	76.5
Deaver (1905-1907)	31	26	83.87
Paterson (collective statistics)	116	109	93.9
Helferich (1905)	56	41	73.3

In Deaver's own cases¹ that were traced, only 58 per cent. had no gastric symptoms after operation, 14 per cent. were markedly improved, 6 per cent. were unimproved, while 14 per cent. had died, either from the original gastric lesion (2 probably from cancer) or from some late complication, such as intestinal obstruction, or vicious circle, indirectly caused by the stomach condition. If we include in the surgical statistics the results of operative treatment administered by surgeons of less experience and skill, the showing is certainly not as much in favor of surgical intervention as we have been led to suppose.

Bettmann and White found that of 126 cases who survived the operation (10 per cent. having died as an immediate result of the operation), and who were under observation for a year or more, only 64.3 per cent. remained well, 6.3 per cent. were much better, while 24.7 per cent. reported as little or no better.

Paterson, by tracing the history of 116 patients who had been operated upon by gastro-enterostomy at periods varying from two to nineteen years, concludes that but 85 per cent. are permanently cured. It is believed by the writer that even under skilled surgical treatment this latter figure or even a little less will be nearer correct than the higher estimates given, that 8 to 10 per cent. will die directly or indirectly from their operation, and that at least an equal number will derive no benefit from their treatment.

W. J. Mayo has made, however, an important distinction between the surgical end-results of ulcers near the pylorus and those of the body of the stomach, the importance of which cannot be overstated.

Basing his conclusions on a series of 1000 cases of ulcer operated on by himself and C. H. Mayo, his conclusions are that the treatment of all duodenal and all obstructing ulcers of the pyloric end of the

¹ Deaver and Ashhurst, *Surgery of the Upper Abdomen*, i, 108.

stomach by gastrojejunostomy and excision or infolding the ulcer gives 98 per cent. of cures or great improvement, while 85 per cent. of ulcers of the body of the stomach will be cured or greatly relieved by excision or devitalizing suture compression with gastrojejunostomy with or without closure of the pylorus. The remaining 15 per cent. were all more or less benefited. None were made worse by the operation. The series was terminated January 17, 1911.

COMPLICATIONS OF ULCER

The Malignant Degeneration of Chronic Ulcers.—The possibility of carcinomatous change in chronic gastric ulcers is of great interest pathologically, and of very great importance from a clinical standpoint. That such changes do occur is well recognized. The frequency of such transitions is more fully discussed on page 223.

The older observations were practically all made upon postmortem material. Such material is notoriously unreliable for such study, as the process is generally so far advanced that all of the earlier changes are obliterated, and one generally finds only a mass of carcinomatous tissue in one or other portion of which there is an ulcer. It is, indeed, often impossible to tell from what portion of the stomach wall the tumor took its origin. There may be one large ulcer with a scar tissue or necrotic base, or several smaller ones, and quite often it seems reasonably certain that these ulcers represent secondary loss of substance in a primary carcinoma.

The more recent investigations have, for the most part, been carried on with specimens removed at operation, and it is to such material that we must turn for a correct appreciation of the pathological changes involved. Nearly all of Wilson and MacCarthy's work was done with this surgical material, and from their extensive observations they believe that it is possible for one to recognize the following changes in sequence.

"1. Chronic ulcers, from the centres of which the mucosa has disappeared, leaving a scar-tissue base.

"2. In the overhanging borders of the ulcers the mucosa is proliferating.

"3. Deep in the borders, many groups of epithelial cells have been nipped off by scar tissue and are exhibiting all stages of aberrant proliferation with infiltration of the surrounding tissues.

"4. Metastases are forming in the lymphatics of the stomach wall and adnexa."

Stages.—Ménétrier, as a result of his studies in 1900, gives his theory of carcinomatous change in chronic ulcers in the following stages (Deaver and Ashhurst):

“First Stage: This is purely inflammatory. There is a chronic gastritis, and the cells lining the glands lose their special and distinctive features (the histological picture is simplified), and the ‘acid’ cells disappear.

“Second Stage: Adenomatous in character. The proliferating glands, deprived of their characteristic elements, become more contorted and convoluted; their cells increase in number; cysts form as a result of obstruction of the gland ducts by proliferation of their lining cells.

“Third Stage: Epitheliomatous in character. The cell groups break through the muscularis mucosæ, and finally are found lying free among the connective tissue of the gastric wall.”

On gross examination of some of the ulcers it is difficult to say whether there has been any malignant change. They appear as ordinary chronic ulcers, with indurated borders, generally showing an abrupt, somewhat undermined margin with overhanging mucosa proximally, with a more gradually sloping wall toward the pylorus. In other ulcers in which such a transition is taking place, the changes are more apparent to the eye. In the first place, these ulcers are apt to be larger. Mayo believes that most ulcers which are larger than a twenty-five cent piece are undergoing malignant degeneration. While they may maintain roughly the form mentioned above, one usually finds more extensive induration about the ulcer, with thickening of the stomach walls, and commonly a definite tumor mass. The ulcer base is generally composed of scar tissue.

Microscopically, the first changes consist of small islands or alveoli of epithelial cells deep down in the mucosa, cut off from the glandular epithelium by more or less inflammatory tissue. These areas are usually found in the margins of the ulcer, and from these isolated islands of cells aberrant and atypical epithelial proliferation may arise.

A little later change shows these isolated groups of cells actively proliferating, but still more or less confined within normal boundaries. In another portion of the same ulcer, or in another ulcer slightly more advanced in its malignant transition, the proliferating cells may be seen pushing their way through the muscularis mucosæ into the submucosa.

From this stage it is but a step to a typical carcinomatous picture of actively proliferating cords and islands of cells invading the surrounding tissues in an atypical and lawless manner. In these well-marked cases, the type of growth may be adenomatous, scirrhus, or

may occasionally show colloid degeneration. Careful examination will almost always reveal changes in both the proximal and distal margins of the ulcer.

Development of Malignant Changes.—Stromeyer,¹ working under Aschoff's direction, has recently reported the results of his investigations upon the association of chronic gastric ulcer and carcinoma. He does not deny the possibility of a carcinoma developing secondarily in the margin of an ulcer. In fact, one of his cases apparently showed such a transition in a very early stage. However, he believes that in the large majority of cases the ulcer represents a secondary process in a primary carcinoma. His material was obtained partly at autopsy and partly at operation.

He finds that in practically all of his cases there is cancer development in both the proximal and distal walls of the ulcer. Superficially, it extends only a few millimeters outward from the ulcer. In the submucosa it is more marked, while in the base of the ulcer it is most extensive and reaches deepest, even involving the greatly thickened serosa. It is upon this arrangement of the carcinomatous tissue that he bases his argument for the secondary origin of the ulcer, his reasoning being as follows:

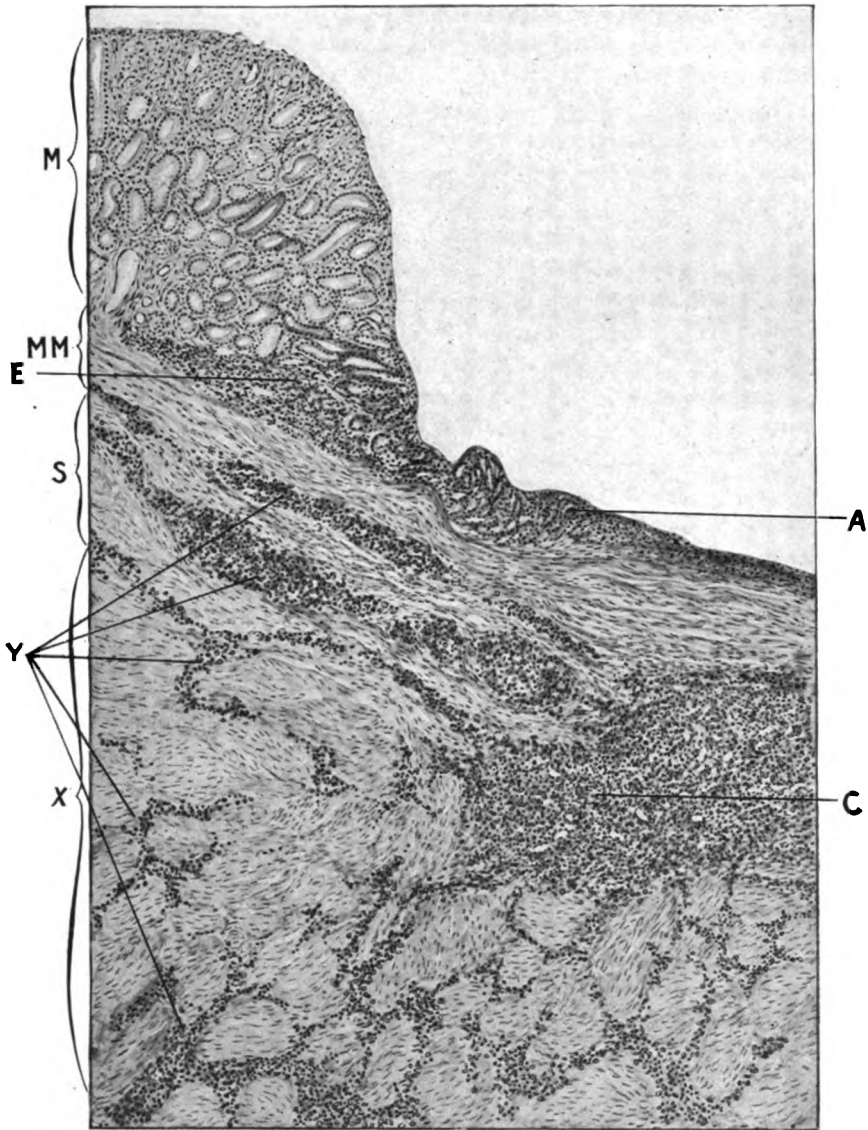
If the cancer development were secondary, one would expect that it had taken its origin from some one point in the mucous membrane of the ulcer margin. One could then easily understand the development of a cancer focus located beside the ulcer, or even encroaching upon it to some extent. But it would be impossible to understand why just the deepest part of the ulcer, far removed from the margins, should be most strongly infiltrated, while the cancer development in the margins should only reach a few millimeters outward. The cicatricial tissue forming the floor of the ulcer would, moreover, offer more unfavorable ground for the infiltration of the cancer than the relatively normal portions of the stomach wall lying adjacent to the ulcer.

This arrangement of the cancer tissue is best explained on the ground that the centre of growth was in the mucous membrane originally present over the centre of the ulcer itself, and that from there the cancer, following Ribbert's laws of growth, extended into the deepest parts of the stomach wall beneath, as well as into the submucosa. The mechanical irritation, softening and disintegration of the mass of cancer tissue, would then lead to the production of an ulcer, with the above-mentioned arrangement of cancer tissue.

It is an undisputed fact that such changes do occur and threaten the life of a certain proportion of ulcer patients. Just how great this

¹ Ziegler's Beiträge, September 12, 1912.

FIG. 28



Carcinomatous ulcer of stomach. *M*, mucosa showing almost perpendicular margin. There is considerable round-cell infiltration at *E*. At *A*, the mucosa is gradually disappearing to be replaced by necrotic tissue; *MM*, muscularis mucosae; *S*, submucosa, containing much connective tissue and strands of carcinoma tissue; *X*, muscularis; *C*, large mass of carcinoma tissue; *Y*, carcinoma infiltrating muscularis in every direction. This drawing shows especially the large amount of carcinoma in the base of the ulcer, with infiltration of the muscularis and marked infiltration along the line of the submucosa as described by Stromeyer.

proportion is, is a matter of some dispute, the figures ranging from the older statistics of 6 per cent. to the present estimate of 71 per cent. of Wilson and MacCarty writing from the Mayo clinic. The writer can only state that in but 7 per cent. of his cancer cases was it possible to obtain a previous history of indigestion that might, even with leniency, be interpreted as due to previous ulceration, neither has he found carcinoma of frequent occurrence in those whom he has treated for ulcer.

Ulcus carcinomatosum may be suspected in a case of supposed simple ulcer under the following conditions:

1. When lactic acid or lactic acid bacilli appear in the gastric contents. The degree of hydrochloric acidity is of much less importance than this. Reductions may occur, but, as a rule, hydrochloric acidity remains high in many cases even throughout the course of the disease. When lactic acid appears it is probable that the patient has passed the stage in which operation can result in radical cure.

2. Cases of uncomplicated ulcer that do not improve under treatment, especially if blood tests of the stools be positive during the third and fourth week of treatment, are to be regarded with suspicion.

3. General failure of flesh and strength following an ulcer cure without apparent cause, with or without occult bleeding, justify exploration.

4. Sudden failure in appetite amounting often to actual aversion to all food, that cannot be explained by temporary derangements of digestion, should call for a most painstaking clinical study of the case.

There is nothing in the physical examination of the patient to assist in forming a correct diagnosis. The only hope of the patient when such a complication occurs, lies in a radical operation, performed so early in the disease that a diagnosis of malignant degeneration cannot be definitely made, at the time at which the operation is advised. In other words, certainty of diagnosis means that it is too late to hope for radical cure—and that to give the patient every possible chance, exploration must be advised and urged in those whose symptoms are merely suspicious of early malignancy. Should exploration be negative, no great harm will result; should it be justified, a life may be saved that otherwise is doomed to a distressing end.

Perforation.—The frequency of perforation is much greater in hospitals as the symptoms of ulceration are often ignored by ignorant patients until they are forced to enter the wards with hemorrhage or perforation.

Habershon¹ places the frequency at 18 per cent., Gerhardt at 13

¹ St. Bartholomew's Hospital Reports, 1890.

per cent., and Cantlie, at the Royal Victoria Hospital, Montreal, at 10 per cent. Fenwick and Deaver consider the accident somewhat less frequent, the former estimating the casualty at 5.5 per cent., the latter at 4 per cent. In the writer's series of all hospital and private cases, 4 per cent. perforated, the complication being six times more frequent in the hospital series than in the private cases. Perforations are less common with ulcers that have previously bled, because of the more radical and efficient treatment that naturally follows the hemorrhage.

Perforation may occur with both acute and chronic ulcer, but is less common with the chronic form, owing to the protective thickening of the base of the ulcer and the adhesions that may form at the site of the lesion, reinforcing the point of weakness.

Ulcers of the anterior wall are more liable to perforate than are those elsewhere located. Pariser estimates that of 200 ulcers, 190 will be on the posterior wall, and of these 4 will perforate, whereas of the 10 ulcers on the anterior wall, perforation would occur in 8. Ashhurst concludes that 8 per cent. of ulcers are located on the anterior wall, and this 8 per cent. furnishes nearly three-fourths of all the perforations.

According to Fenwick, the acute gastric ulcer usually perforates the comparatively thin wall of the stomach in the cardiac half of the viscus on the anterior wall near the lesser curvature, whereas the chronic form is most prone to perforate in the pyloric portion of the stomach on the posterior aspect near the upper margin. Perforating ulcers formerly considered gastric are now generally conceded to be in most cases on the duodenal side of the pylorus.

The reason for the greater tendency of ulcers of the anterior wall to perforate lies in the relation of the stomach to surrounding viscera. The anterior wall lies freely exposed to the general peritoneal cavity, and is subject to greater degrees of contraction and dilatation. It is furthermore subject to traumatism acting throughout the anterior abdominal wall.

The posterior surface of the stomach is placed in contact with the rigid protective spinal column within the confines of the lesser peritoneal sac, and is in close relation with the pancreas, duodenum, and liver. Owing to these relations, ulcers on the posterior wall are more apt to contract protecting adhesions and are therefore not so liable to perforate.

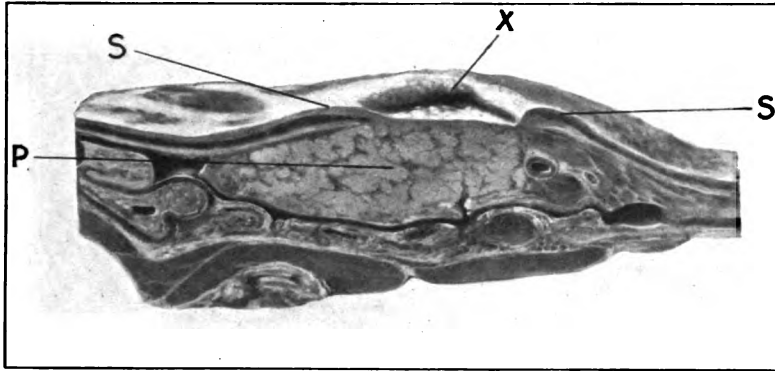
From ulcer of the anterior wall of the duodenum fatal perforation often ensues, owing to the absence of any solid viscus that can act as a secondary basis for the ulcer, but when ulceration occurs on the posterior wall, perforation is often prevented by the pancreas which lie immediately behind it. Were it not for the presence of the pancreas

at this exact point it is probable that a much greater number of duodenal ulcers would perforate.

Recurring perforations in the same individual are not uncommon; in the literature of the year instances of successive operations were reported by Cuff,¹ Willis,² and Schmitzler.³

Perforation may be acute, subacute, or chronic.

FIG. 29



Perforated gastric ulcer. The stomach is adherent to the pancreas which forms the base of the ulcer. X, ulcer; P, pancreas; S, wall of stomach. (From the Pathological Museum of the Presbyterian Hospital, New York.)

Acute Perforation.—In the acute form the perforation is unprotected by adhesions, so that the gastric or duodenal contents escape rapidly and freely into the peritoneal cavity. Following perforation of a gastric ulcer, an acute diffuse peritonitis is at once set up, the infection spreading rapidly to all parts of the abdominal cavity. The contents of the duodenum, on the other hand, are relatively sterile, which accounts for the fact that while the number of the duodenal perforations is large, the death rate is comparatively low. Not only is the duodenum more often empty at the time of the perforation than is the stomach, but a protective spasm of the pylorus is induced at the time of the accident, preventing further ingress of chyme, so that in duodenal perforations the leakage is apt to be limited.

Owing to the anatomical situation of the duodenum, deeply placed between the right lobe of the liver and the gall-bladder, the contents do not, as a rule, escape into the peritoneal cavity as is the case with perforation of a gastric ulcer, but tend to travel along the posterior aspect of the hepatic colon, gravitating downward in front of the right kidney and eventually into the right iliac fossa, simulating renal or

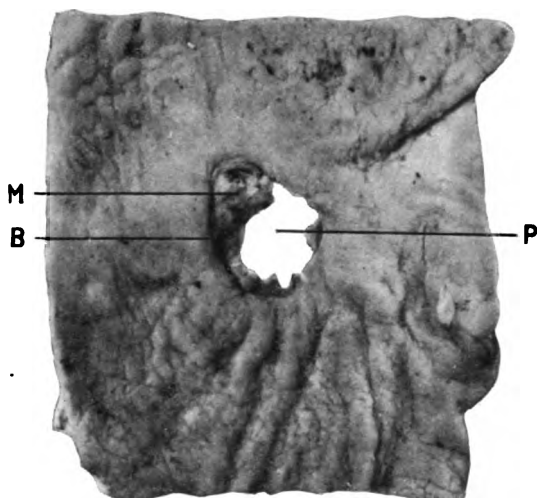
¹ British Medical Journal, 1907, i, 255.

² Ibid., i, 926.

³ Report to Vienna Medical Society, December, 1907.

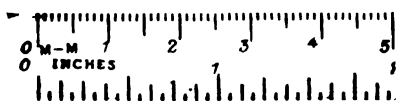
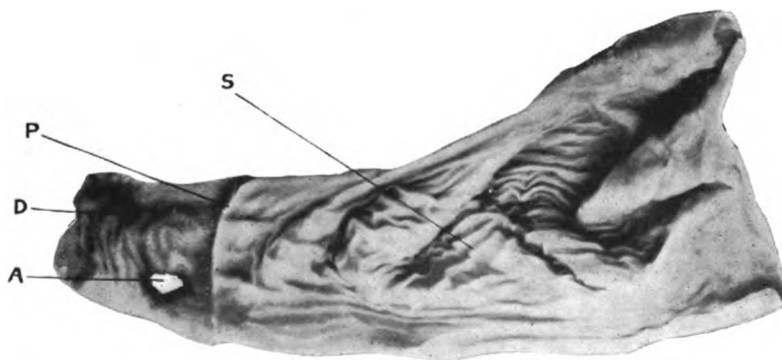
appendicular abscess. This line of infection may occur in the acute form of perforation, although it is especially apt to be the case with perforations that are subacute.

FIG. 30



Perforated gastric ulcer. *P*, perforation; *M*, muscularis, forming what remains as floor of the ulcer; *B*, border of ulcer. (From the Pathological Museum of the Mt. Sinai Hospital, New York.)

FIG. 31



Perforated duodenal ulcer in an infant four months old. *S*, stomach; *D*, duodenum; *P*, pyloric ring; *A*, perforated ulcer with hemorrhagic border. (From the Pathological Museum, Presbyterian Hospital New York.)

Symptoms.—In many of the cases perforation is the first indication of serious gastric trouble, occurring as an initial symptom in 3.4 per cent. of Fenwick's acute ulcers, and in 5 per cent. of the writer's. These seem to be low figures when we realize how many acute perforations are brought to the hospital oblivious of all symptoms of previous ulceration.

Hartley, in a verbal communication to the writer, states that in his last 10 cases of perforation, 8 claimed to be free from pain or indigestion before the accident. In 8 of 13 cases of perforation reported by Andrews a history of previous pain was denied. Many of the patients are, however, either too ill or too stupid to remember or try to describe their symptoms preceding the perforation, but a careful history taken after their recovery will usually elicit the fact that they suffered from more or less epigastric pain before the occurrence of the accident.

Ulcers on the anterior wall are so often unaccompanied by characteristic symptoms that the ulcer is frequently unrecognized until the event of perforation. In other cases perforation is preceded by signs of active ulceration. Increase in the intensity of pain, together with an increase in its constancy, and a localized feeling of "soreness" in the upper right quadrant of the abdomen, made worse on exercise, should occasion apprehension of impending disaster, if these symptoms occur in a patient who is known to have duodenal ulcer.

In duodenal perforation it is usually the chronic form that perforates, which, with but few exceptions, has given sustained evidence of its presence, together with a distinct exacerbation of its symptoms for some days or weeks prior to its rupture.

The accident may occur at any time, even during the hours of sleep; in other cases it may follow the distention of the stomach by food, dietetic indiscretions, straining efforts of the abdominal muscles or external traumatism acting throughout the anterior abdominal wall. It has unfortunately followed the indiscreet use of a stomach-tube and attempts to empty the stomach by emetics.

The symptoms of acute perforation appear simultaneously with the rupture. The patient will suddenly be seized with a pain that is difficult to describe except in terms of severity. It is sharp and agonizing, and even speaking produces additional suffering. The pain is at first referred to the upper part of the abdomen, more usually in the epigastric or the right hypochondriac region, or it may be umbilical. There may be the feeling that something has torn or burst within the abdomen.

Vomiting closely follows the pain in about one-half the cases, the vomited matters being frequently blood-tinged, but repeated vomiting does not seem to occur—an important point in the differential

diagnosis between ulcer and acute intestinal obstruction or strangulation of the bowel, in which repeated efforts of vomiting are made. In other cases vomiting is absent, owing to inhibition of the emetic centres by the shock.

Symptoms of collapse speedily supervene if general peritoneal sepsis results, and are most marked in the perforation of gastric ulcer. In duodenal perforation the shock may be quite insignificant at the onset, may not appear for some hours, or may be altogether absent. The absence of definite symptoms of shock should therefore never deter us from early exploration when perforation is suspected. Syncope may precede the accident shock. Fenwick and Crisp report cases in which syncope followed by shock were the only symptoms present.

The pulse may be rapid and feeble, as is usual in shock, but *in many cases the pulse is slow, regular, and of good force*. This is especially the case with duodenal perforation. Many operations have been delayed too long owing to the general excellence of the pulse, although it is clear that perforation has occurred.

The breathing is generally shallow and thoracic, the movements of the abdomen in respiration are usually restricted, especially on the right side.

The temperature at first may be slightly subnormal, subsequently rising a little, attaining the height of 101° or even more. The chief characteristic of the temperature is its slight elevation compared with the serious condition of the patient.

Leukocytosis is usually well-marked, counts of 12,000 to 30,000 within a few hours of the accident not being uncommon. The polymorphonuclears are regularly increased. In cases of overwhelming infection the total number of leukocytes may not be increased, although there is always a relative increase in the percentages of the polymorphonuclears.

The most constant sign of perforative peritonitis is rigidity. The abdominal wall assumes a board-like rigidity that is most characteristic. Moynihan writes: "No other condition than perforation of the stomach or duodenum can give rise to such unalterable and unyielding tension of the muscles." It never for one instant slackens until death is imminent. The maximum point of rigidity is usually near the site of the perforation, and for this reason early and careful examination should be made to determine the probable site of rupture. It is usually found over the upper part of the right rectus muscle. In duodenal perforation initial rigidity may be slight, occasionally barely to be detected, or there may be an initial rigidity which after a short time grows less so that one may be deceived by a period of apparent improvement. Sooner or later the rigidity reappears and becomes

progressive. In almost every instance of duodenal perforation the right side is not only more rigid but more tender than the left, but the right iliac fossa may be the most exquisite tender area of all. In 49 cases of ruptured duodenal ulcer reported by Moynihan in no fewer than 19 had the diagnosis of appendicitis been made. In rare cases with overwhelming infection death may occur from peritoneal sepsis, without any attempt at repair by plastic exudation. In these cases rigidity may never occur.

Tenderness at first corresponds to the area of greatest pain, later, when peritonitis develops, the tenderness becomes more general. This persists after peritonitis with its consequent tympany has rendered the detection of rigidity difficult. There is a remarkable correspondence between the seat of maximum tenderness and the point of perforation which is of importance in determining the selective point for operation.

Attention has been called to the frequent absence of liver dulness, occasioned by the presence of air or gas in the abdominal cavity, as a sign of perforation. This sign is now recognized as variable, and not distinctive. Tympany without perforation may produce similar physical signs. It should never be relied upon to determine the question of operation.

In 13 of Andrews' cases liver dulness was absent in 3, diminished in 7, and normal in 3.

Of 43 perforation cases compiled by English, liver dulness was absent in 12, diminished in 20, normal in 11.

The fatality varies. In the modern statistics the mortality is much less than in those compiled a few years ago, not only because the importance of early operation is now universally recognized, but also an increasing number of "appendicitis" cases are being sent to the hospitals for operation, and many of these turn out to be perforations. Martens,¹ with a large experience in operating upon gastric perforations, reports that almost without exception his cases were admitted to the hospital with the diagnosis of appendicitis. This is especially apt to be the case in the perforations of duodenal ulcer. According to Deaver, perforation constitutes 80 per cent. of the deaths from ulcer.

Subacute Perforation.—Perforation is said to be subacute in those cases in which the extravasation is so slight that the resulting peritonitis is localized.

Extravasation may be limited from a variety of causes:

1. Whenever ulceration is gradual so that perigastritis occurs, the point of perforation may be protected by adhesions, so that escape of

¹ Deutsch. med. Woch., 1907, p. 1851.

gastric or duodenal contents into the general peritoneal cavity is prevented.

2. The perforation may occur at a time when the viscus is empty. This fortunate circumstance is more common in duodenal than in gastric ulcer, for obvious reasons. In rupture of duodenal ulcer the protective closure of the pylorus preventing escape of contents of stomach into the ruptured portion, has previously been alluded to.

3. The rent may be more or less completely closed by an exudation of plastic lymph thrown out just before the time of actual rupture, or else, in rare instances, the omentum may be found plugging up the false opening.

When local peritonitis occurs in subacute perforation there is formed an abscess cavity communicating with the stomach or duodenum and shut off by adhesions from the general peritoneal sac. Such an abscess may remain for a considerable period of time without marked change, draining through the point of rupture, or else it may extend, the rule being that extension occurs more readily in the direction of that part of the wall that is formed by adhesions. In many cases this advancing infection leads to a progressive fibrinopurulent peritonitis. In other cases the abscess may invade the structure of the liver, more rarely the substances of the pancreas, or there may be formed fistulous openings into hollow viscera, or through the anterior abdominal wall. Perforation of aorta, portal vein, and superior mesenteric vein has occurred. Gastrocolic fistula is, however, less common in ulcer than in cancer. Many interesting cases are described showing to what a remarkable extent burrowing of the pus has occurred. A case has been reported of the burrowing of the abscess along the greater vessels of the neck when it pointed and discharged. In one case the pus was discharged at the angle of the right scapula. It is clear that an abscess which forms as the result of subacute perforation may burrow in any direction, and may reach the body in any region by almost any route, however devious it may appear.

The most common form of abscess in this form of perforation is the type commonly spoken of as subphrenic abscess. Abscesses caused by rupture of anterior ulcers or of ulcers near the lesser curvature usually form under the left lobe of the liver, being bounded by the stomach below, and posteriorly by the gastric hepatic omentum. If rupture occurs of an ulcer near the fundus the left boundary may be formed by the spleen.

Perforation of ulcers on the posterior wall usually cause an infection of the lesser peritoneal cavity (an "empyema of the bursa omentalis"). The purulent process frequently invades the substance of the liver, or the pus may burrow in front of the anterior margin of the left lobe

of the liver, so as to be subdiaphragmatic in type. Infection of the pleura is common in these cases, usually resulting in empyema. Perforation into the pleura, lung, or pericardium may occur. Pneumonia is not uncommon.

Symptoms.—The symptoms of subacute perforation differ from those of the acute form chiefly in their degree of intensity. An increase of ulcer symptoms is usually noted preceding the perforation—pain more intense, more constant, and more readily increased by exertion. The feeling of local soreness and an increased tenderness are suspicious signs of impending disaster, and in an ulcer patient should always excite apprehension.

The initial pain is severe, often excessively so, but it lacks the agonizing quality seen in the acute cases. The pain may lessen considerably in a few hours, often deceiving us as to the gravity of the attack. The pain is usually more accurately localized than in the acute cases.

Shock is less frequent than in the acute form, amounting in the majority of cases merely to a sense of extreme prostration and a realization of serious illness. Vomiting is somewhat more common. The appearance of the patient may be quite normal. The abdomen may be everywhere tender, but the point of maximum tenderness is usually quite distinct. Rigidity is regularly present, but more localized and less general than in the acute types. Moynihan describes the painful and tender area feeling "as if a flat, hard disk had been inserted into the abdominal wall." The symptoms change slowly. The tenderness may become more localized, or may spread, if progredient peritonitis result. Subsequent symptoms are those of sepsis, common to all suppurative foci.

Chronic Perforation.—A perforation may be said to be chronic when by reason of perigastric adhesions no further infection of peritoneum either local or general ensues. In the majority of these instances the base of the ulcer has become adherent to some of the neighboring organs, so that by an extension of the ulcerative process, the base of the ulcer is formed by pancreatic or hepatic tissue, or else by dense fibrinous material. Many patients go on for years with an ulcer whose base is formed by one of the above-mentioned structures. In other cases extension into these adherent structures may occur, causing abscess cavities and fistulous tracts. The time at which the actual penetration of the true ulcer base occurs is not apt to be marked by characteristic symptoms. Pain more or less constant and influenced unfavorably by exertion and by the dragging of the stomach by the mechanical weight of food, are the chief symptoms. Pus is present in the fasting stomach of these patients, and is of the utmost importance in diagnosis. The stomach contents are not, however, foul, as is so

frequently the case of progressive ulceration of neighboring organs in cancer.

Tenderness is persistent and localized. Local rigidity is usually, but not invariably present. Symptoms of sepsis induced by slow absorption, progressive emaciation, fever of an intermittent type, often accompanied by chills and sweating, are liable to supervene.

Differential Diagnosis of Perforation.—In the majority of instances the occurrence of perforation may be readily diagnosticated, although it may be difficult, in the absence of previous history, to determine the site of perforation. The sudden onset, tendency to collapse, and, most important, the presence of unyielding, unchanging rigidity, make it evident that an acute surgical abdomen exists. In the majority of hospital patients a finer diagnosis than this is not of much value, nor is it important to differentiate between gastric and duodenal perforations. The main thing is to decide that a perforation has taken place, and to operate without further delay. The seat of maximum tenderness and the initial area of rigidity or the area of greatest rigidity are sufficient to determine the seat of incision.

Mistakes, however, have been frequently made, even by the most careful and experienced surgeons. The abdomen has been frequently opened for perforation, and no perforation found.

Manges reports the case of a woman, aged twenty-seven years, with the clinical history of gastric ulcer, who suddenly was seized by intense pain and tenderness in epigastrium with marked rigidity. Operation was performed, but no perforation and no peritonitis found—merely an uncomplicated gastric ulcer.

Diagnosis from Pleurisy and Acute Lobar Pneumonia.—Pleurisy, especially diaphragmatic, and acute lobar pneumonia may be accompanied at the onset by acute severe pain in the abdomen and abdominal rigidity, so closely simulating perforation as to deceive even the most expert surgeons.

In the pulmonary cases, however, the temperature is usually high, ranging between 103° and 105°, whereas in perforation the temperature exceeds the height of 102° only in rare instances. In the cases of pulmonary origin the respirations are frequent, usually 30 to 40, which, accompanied at the onset by a pulse not usually over 100, is quite different from the rapid pulse and but slightly accelerated respiration rate of perforation. In the pulmonary cases abdominal rigidity is neither as well-marked nor as unchanging as in perforation—it is not uncommon to notice differences in its intensity at different times during the physical examination. Especially apt is the rigidity to yield for an instant, at the end of expiration. This does not occur with ulcer.

Diagnosis from Erythematous Diseases.—In the various gastro-intestinal crises of the erythema group of skin diseases we may have a symptom-complex somewhat misleading, but we are aided in our diagnosis by the history of previous attacks, by the spasmodic colicky character of the pain, and by the evidences of erythema, purpura, angioneurotic edema or urticaria of the skin. Nevertheless the abdomen has been opened under an erroneous diagnosis in these cases.

Diagnosis from Appendicitis.—The disease most frequently mistaken for perforation is appendicitis. The frequency in which perforated ulcers, especially of the duodenum, enter the hospitals with the diagnosis of appendicitis, has already been attended to.

In some cases a differential diagnosis is a matter of extreme difficulty, although the consequences are not serious as long as the condition of acute surgical abdomen is recognized, although in the majority of cases the diagnosis may be made. The various points for diagnosis are shown in the accompanying table.

DIFFERENTIAL DIAGNOSIS BETWEEN PERFORATION OF ULCER AND APPENDICITIS

<i>Perforated Ulcer</i>	<i>Perforated Appendix</i>
Pain sudden and overwhelming.	Pain more gradual and more colicky.
Pain referred to upper abdomen, gradually becoming general.	Pain general at first, becoming gradually more localized.
Collapse marked.	Collapse usually slight.
Rigidity general, intense.	Rigidity localized, less unyielding.

Diagnosis from Acute Hemorrhagic Pancreatitis.—Acute hemorrhagic pancreatitis may resemble perforation in the severity of the initial pain and consequent shock.

The previous history, if any is obtained, is more often that of gallstones—inaugural symptoms of ulcer are not obtained. The pulse is bad from the start and disproportionate to all other vital signs. The face is usually slightly cyanosed. Vomiting is a more conspicuous feature. Tenderness is most marked in the median line over the inflamed gland—not more intense on the right side as is the case in the majority of perforations. A painful, tender, tumor mass may even be felt, not moving with respiration, and lying transversely just above the navel. Rigidity is usually less marked and less general in pancreatitis than in perforation.

Despite these points of difference, a differential diagnosis between the two conditions may be impossible, especially when posterior perforations have involved the lesser peritoneal cavity forming an empyema of that sac.

Diagnosis from Acute Intestinal Obstruction.—Acute intestinal obstruction is marked by the onset of acute, severe pain and by ab-

dominal rigidity. The pain, however, is more wave-like and colicky in character—initial collapse is usually wanting. Rigidity is not as marked as in perforation, and often is delayed until the occurrence of peritonitis which regularly appears late in these cases. The temperature is not elevated until the appearance of peritonitis. The most marked symptom of obstruction is vomiting—not only of ingested food, but of intestinal contents approaching the stercoraceous in type. Vomiting, on the other hand, occurs only in about one-half of the cases of perforation, and the vomiting act is not apt to be repeated.

Rupture of the Gall-bladder.—Rupture of the gall-bladder seldom occurs with the suddenness of perforation. There are usually marked local symptoms of severe gall-bladder infection preceding the rupture, although cases have been reported in which a differential diagnosis before the operation has been impossible.

Perigastritis and Adhesions.—Ulcers which encroach upon the peritoneal coat of the stomach are regularly accompanied by local thickening of the peritoneum at the site of their base. This is a conservative process, as it reinforces the weakened area and prevents perforation. In many cases the stomach becomes adherent at this point to any one of the neighboring organs. These adhesions differ in no respect from those encountered in other parts of the peritoneal cavity, although owing to the chronicity of the ulcer they are more apt to be dense and resistant. Occasionally there occurs a deposition of lime salts in these adhesions sufficient to turn the edge of a knife.

Adhesions with neighboring parts are contracted with less frequency than was at one time supposed. Jaksch found adhesions in 40 per cent. of his cases, Lebert in 42 per cent., while in Fenwick's cases of chronic ulceration they were present in 46 per cent. On the other hand, Fenwick found adhesions in 6 per cent. of his acute cases. To show the relative frequency with which the various neighboring organs become adherent to the bases of the ulcer, Fenwick gives the following table, compiled from an analysis of 123 cases:

Organ.	Number of cases adherent.	Per cent.
Pancreas alone	49	40.0
Liver alone	30	25.8
Pancreas and liver	10	8.1
Colon	7	5.7
Liver and colon	4	3.2
Spleen	2	1.6
Mesentery	3	2.4
Three or more organs	15	12.2
		<hr/>
		100.0 100.0

The ulcer thus contracts adhesions with the liver and pancreas in three-fourths of all the cases in which adhesions are present.

Although a local perigastritis is nature's method of putting a patch upon the weakened point, the presence of adhesions is not without certain deleterious results. The fixation of the movable stomach upon an immovable organ such as the liver or pancreas naturally tends to hamper the muscular movements of the stomach wall, and to lead to more or less muscular insufficiency. Furthermore, such a fixation may seriously interfere with the proper contraction of the ulcer that is necessary for its repair. Again, many deaths occur from hemorrhage the result of adhesions to and erosions into the neighboring parts; the bloodvessels of the pancreas and liver have frequently been opened and erosions into almost all of the neighboring bloodvessels has occurred.

In the majority of instances, perigastric adhesions give no additional symptoms to those of the ulcer itself. In other cases the adhesions are responsible for many obscure symptoms which develop after the cure of chronic ulcer, although they seldom seem to interfere with the processes of digestion, or to shorten life. These symptoms may be of neuralgic character occurring from time to time without any direct relation to the taking of food, or they may appear after eating, usually within a very short time after the meal, as a result of the mechanical interference with the movements of the stomach, and are proportionate to the quantity rather than to the quality of the food. In other cases they occasion only a sense of vague uneasiness more or less constant, or an annoying sensation described by the sufferers as a "dragging" pain. It is very characteristic of all localized inflammation of serous membranes that adhesions give rise to a pain which is increased by exertion and by an erect posture. The patients say that their pain diminishes during the night and gradually increases during the day, to arrive at its maximum during the early evening, and that they obtain relief from their pain only by lying down. The increase in these pains by the mechanical weight of food is indicative of a gastric origin.

During the acute exacerbation of perigastric inflammation, the pain becomes more or less constant and is influenced to a greater extent by exertion, by change in posture, and by the mechanical dragging whenever food or drink is taken. The increase in local tenderness, and usually a localized rigidity, accompany these exacerbations.

Pain in the region of the shoulder is commonly observed when there are adhesions binding the stomach to the liver. Steady boring pains in the back occurring in attacks of great severity are suggestive of adhesions to the pancreas, with or without chronic perforation and erosion of its substance. In the latter instance pus cells are often

found in the gastric contents. Pain running around the chest and causing a feeling of constriction in the lower thorax is observed in ulcers of the lesser curvature with adhesions to the liver or diaphragm especially if these ulcers are near the cardiac end of the stomach. Duodenal ulcers frequently contract adhesions with the gall-bladder.

In ulcers of the first portion or at the junction of the first and second portions of the duodenum, on its upper posterior wall, the common bile duct may be caught by adhesions as it passes behind the duodenum, and compressed. A number of such cases have been recorded. Portal obstruction or portal thrombosis from compression has occurred as a result of deep cicatrization of a chronic duodenal ulcer.

Progressive Perigastritis.—It may happen that the perigastritis instead of remaining localized to the site of the ulcer, may spread so as to involve a greater part of the surface of the stomach. This form of inflammation is very chronic in its type, leads to a very marked thickening of the peritoneal coat of the stomach, and even extends to involve the peritoneal folds which connect the stomach with the neighboring parts. In some instances the stomach is so firmly welded to all the structures about it that its dissection is impossible. The result is that the peristaltic movements of the stomach are greatly impaired. By contraction of the fibrous thickening of its wall the stomach may become compressed and distorted, being occasionally reduced to the size and shape of a section of the bowel. Fibrous cords may form, pass across the anterior surface of the stomach, usually between the liver and the great omentum, which may divide the stomach into two unequal pouches, practically forming the hour-glass stomach. In some cases there results a line of ulceration in the mucous membrane along the line of compression. Volvulus may occur in these cases.

Symptoms.—The symptoms of this form of perigastritis are somewhat indefinite in character, many cases running their course without any symptoms whatever. In other cases there is a marked intolerance of the stomach for any ordinary quantity of food, and the stomach will reject its contents if more than a small, definite amount be taken. Ordinarily there is pain, more or less constant, in the upper abdomen, with tenderness which often passes beyond the normal stomach boundaries. This extension of the tenderness is due to the involvement of the great or lesser omentum. Vomiting and exacerbation are attendant symptoms. Edema and ascites may occur from compression of the portal vein. Slightly irregular temperature is noted in a majority of cases.

Cicatricial Contractions.—Pyloric stenosis is the commonest sequela of gastric ulcer. In almost all ulcers at or near the pylorus there occurs

a slight impairment of the pyloric opening, which gives rise to the hyperacidity and hypersecretion to which reference has previously been made. Temporary pyloric spasm characterized by attacks of acute hypersecretion is common during the course of both acute and chronic ulcer. These two forms of pyloric contraction are very slight and often of but short duration, and it is not customary to speak of either one as pyloric stenosis. This term should be reserved for those cases in which cicatricial contraction has occurred at the site of the ulcer, causing a definite and permanent contraction at the pyloric orifice. Its greatest distinctive feature is the constant presence of food remains in the fasting stomach. These cases are described in detail under the heading of Benign Pyloric Stenosis.

Stenosis of the cardiac end of the stomach may occur as the result of cicatricial contraction at that point. But cases of cardiospasm are frequently observed in which muscular contraction at the cardiac orifice continues without abatement for years after the healing of the ulcer in this situation, even though cicatrized tissue has not formed at this point. It is probable that the majority of cases of cardiospasm originate in this way. Stricture of the duodenum may occur. The contracting band may be thin as whipcord, narrowing the bowel as if a string had been tied around it, or the contraction may be long and tortuous, forming an hour-glass contraction of the duodenum. W. J. Mayo and Moynihan each report a case of hour-glass contraction of the stomach and of the duodenum in the same patient. The symptoms of obstruction of the duodenum resemble those of pyloric stenosis. If the contraction be above the papilla a differential diagnosis is practically impossible. If the contraction be below the papilla a diagnosis is made by the constant presence of food remains, together with fresh bile in the fasting stomach. In the gastric contents it may furthermore be possible to obtain pancreatic reactions. Stricture of the duodenum near or around the ampulla of Vater may involve both the common bile duct and the canal of Wirsung. Acute or chronic pancreatitis may result. Jaundice and inanition may ensue, and a suspicion of malignant disease of the pancreas may be entertained.

Hour-glass Contraction.—The hour-glass contraction of the stomach, like pyloric stenosis, is rather a result than a complication of gastric ulcer. The stomach may be so deformed by contracting bands as to be contracted in its middle zone so as to form two stomach pouches, separated by a contracted opening which varies greatly in size. A similar contraction of the stomach into lateral pouches occurs in those cases of peritoneal bands, which cross the stomach and form a line of compression. Allusion has just been made to these cases. By multiple contractions the stomach may be trilocular. Moynihan has described

a case in which by multiplicity of bands the stomach was divided into four separate sacs.

Saddle-back ulcers on the lesser curvature are more frequently followed by hour-glass contraction than are any other form. The symptoms, physical signs, and means employed in diagnosis are described under separate headings.

Phlegmonous Gastritis.—Of 91 cases of phlegmonous gastritis collected by Schnarrwyler, with additions by Robertson, gastric ulcer was by far the most common associated lesion, being present in 17 of the cases.

TREATMENT

General Measures.—Rest and protection of the stomach from all mechanical, chemical, and thermal insults are the indications for the medical treatment of ulcers. These requirements are complied with by bodily rest and a carefully selected diet, usually preceded by a short period of total abstinence from all food and drink.

Bodily Rest.—Bodily rest is almost essential in the treatment of ulcer, not only because the mechanical agitation and dragging of the ulcer by the weight of the food during locomotion and even as the result of the upright posture, are prevented, but because we can begin our diet by giving scanty quantities of food and drink for the purpose of throwing the minimum amount of work upon the stomach, quantities at first so insufficient that the patient would be unable to be up and about without unpleasant or even serious results.

The patient should be kept in bed from two to four weeks, in ordinary cases. In those whose symptoms are rebellious to treatment, a longer time is required. Quiet and mental repose are essential, and it is occasionally better to postpone the treatment until the patient can arrange his affairs so as to enter upon the cure with tranquillity, provided too long a time is not demanded, and that the patient in the meantime does not suffer in consequence. As a rule, however, the sooner the treatment is begun after the diagnosis has been made, the better are the chances for ultimate recovery. *Any treatment for ulcer that does not include absolute rest in bed is but a half-hearted treatment, and will obtain only partial results.* No delay should be permitted in cases with hemorrhages, either visible or occult, or in those patients whose increase of pain and tenderness suggest the possibility of impending disaster. If hemorrhage be recent, the rest must be absolute; and the rising even for toilet purposes be strictly forbidden.

External Applications.—External applications are to be employed during the first few weeks of treatment.

In cases with hemorrhage, either visible or occult, ice-bags should be applied constantly to the epigastrium, so as to promote firm contraction of the bleeding viscus. Under no circumstances should hot applications be applied, as it has been proved conclusively that the application of external heat increases the congestion of the stomach, and is frequently followed by a recurrence of the hemorrhage. The ice-bags should be applied until all traces of blood have disappeared from the stools.

In cases without hemorrhage moist applications are to be employed, as hot as can be given or endured. Hot poultices or flannel or spongipiline wrung out of hot water, and applied every half-hour, may be used, and are especially recommended by von Leube, who insists that retardation of the healing process inevitably results when less extreme degrees of heat are employed. Very convenient is the use of the electric pad, because the degree of heat can be easily regulated, and the number of attendants diminished. A piece of moistened flannel should be placed under the pad, and the whole tightly girded by an abdominal binder.

Lesser degrees of heat may be given by the Priessnitz compress. A layer of flannel or a piece of spongioline about ten inches square is wrung out of hot water and covered with mosetig batiste or oiled silk, over which is placed a folded towel, large enough to overlap the edges of the cataplasm. A snug abdominal binder is then applied. If the flannel be not wrung dry enough, it will drip and leak upon the clothes and bedding. If the binder be not tightly applied, air will get up under it and cause chilling of the surface. The pad need not be changed oftener than every two to four hours in the day and once in the night. It is not, however, as effective in cure as the more extreme degrees of temperature.

Abstinence.—In cases with recent hemorrhage, either visible or occult, it is usual to insist upon total abstinence from all food and drink, so as to place the stomach in the condition of complete physiological rest, and this abstinence is to be continued until all traces of blood have disappeared from the stools. Usually this occurs by the third day, but although the hemorrhage may continue longer than this, it is rarely advisable to prolong the abstinence period for more than five days, although in extreme cases one is to be guided by the general condition of the patient. The abstinence must be absolute; not even cracked ice should be allowed. It is the custom of many to begin the ulcer cure by a period of abstinence, even in those cases that are not attended by hemorrhage.

During the period of abstinence, there is much less discomfort experienced than one would imagine possible. The chief annoyance is thirst,

but by the use of constant mouth washes, this disagreeable symptom is rendered quite endurable. The sanitary care of the mouth is, moreover, of great importance in minimizing the danger of parotid infection.

Nutritive Enemas.—Nutritive enemas are frequently employed during the period of starvation. The tube should not be inserted more than four inches, and the fluid should be allowed to enter slowly and under low pressure, stopping if pain or tenesmus should occur. Never more than 10 ounces should be used at any one time, and not more than three or four injections are to be given daily. The temperature must be that of body heat. To facilitate retention, the pelvis may be elevated by pillows, and in all cases absolute quiet must be enforced following the injection.

Solutions of peptone (10 to 15 per cent.), egg-albumen, peptonized milk, sugar solutions, or boiled starch may be used—the addition of a small quantity of salt seems to favor absorption. A few drops of laudanum or of the deodorized tincture of opium may be added should the bowel be irritable.

Ewald recommends an enema composed of two tablespoonfuls of flour boiled with 150 c.c. of water or milk, to which are added one or two eggs, 50 to 100 c.c. of a 15 to 20 per cent. solution of glucose, and a knife-point of salt.

Boas' enema consists of 250 c.c. of milk, the yolks of two eggs, one tablespoonful of red wine, one tablespoonful of Kraftmehl, a knife-point of salt, and 5 drops of tincture of opium.

The writer believes that the value of nutrient enemas is more from the quantity of liquid introduced than from the caloric value of the ingredients, as but little of nutrient value is absorbed. Albumin and sugar solutions usually add to the putrefactive processes in the bowel. He further believes that unless enemas are called for to combat depressant symptoms, they do more harm than good, by exciting peristalsis which we are most anxious to avoid. He, therefore, recommends that for an abstinent period of from three to five days, enemas of nutritive value be not given, or any rectal injections of any kind unless thirst be excessive or depressant effects of the abstinence become evident. Under these circumstances there may be given injections of decinormal solutions of sodium bicarbonate, in preference to salt solution usually employed, for the reason that it more effectually controls thirst and combats any tendency to acidosis from the enforced starvation.

The continuous instillation of the soda solution is preferable to the use of enemas, using any of the modifications of the Murphy drip. In this way water at the rate of a pint an hour can be introduced without discomfort and without exciting peristalsis.

Diet.—Various diets have been recommended in the treatment of ulcer, both during the actual ulcer cure, and also during the period of convalescence. There are certain characteristics in all these forms of diet which must be insisted upon.

The diet must be sufficient in nutritive value to prevent emaciation.

In all forms of diet given during the first and second week of treatment, the nutritive value is not sufficient to maintain body weight, and there usually occurs loss of from six to ten pounds. During the third and fourth weeks the diet is usually increased; so that a great portion of this loss is recovered. After the fourth week it is of importance to maintain this body weight, without further loss, no matter what form of diet is employed. Prolonged administration of milk is unsuitable, because at least four liters must be given daily to bring the caloric value up to the requisite point.

It is a recognized clinical fact that the healing of an ulcer is regularly retarded by anemic and depressed conditions of the patient's vitality, and therefore it becomes a question of great nicety how far the diet can be carried without detriment to the patient's ultimate chances of recovery. Of course, in deciding this point, much depends upon the general condition of the patient at the time when the treatment is begun. One would naturally not starve an anemic and depleted patient to the same extent that would be justifiable in one who is robust and plethoric.

The Daily Quantity of Food Given Must Not Be Excessive.—One of the objects of the treatment of ulcer is to allow the stomach to contract, as the healing process proceeds more rapidly under these circumstances.

Fleiner writes that by a concentrated diet "we render it possible for the stomach to shrink to its smallest compass. The edges of the ulcer then tend to approach each other, and the deepest portion of the ulceration approaches the level of the edges, so that the whole ulcer becomes smaller and more shallow."

A diet exclusively of milk is unsuitable, as distention of the stomach is bound to occur if enough in quantity is given to yield sufficient nourishment to the patient, and to maintain bodily strength.

Nourishment Must be Non-irritating and Bland.—Coarse and irritating articles of food do not as rapidly pass through the pylorus as does food devoid of all irritating qualities, and the longer the food remains in the stomach, the greater tendency toward hypersecretion and hyperacidity. It is, moreover, obvious, that by unsuitable diet, we add to the irritation of the ulcer and prevent its healing.

The Diet Should Be Such as to Successfully Combat Hyperacidity.—With this object in view, two different forms of diet are in vogue,

one, indorsed by von Leube and the majority of clinicians, comprises bland articles of food, chiefly carbohydrates, which require the minimum gastric secretion, the other, known as the Lenhartz diet, is composed of concentrated albuminous foods that unite well with hydrochloric acid in the gastric juice to form a non-irritating acid albumin. These two diets are later given in detail.

Medical Treatment.—*The medical treatment of ulcer* may be divided (1) into that which is given as a more or less routine, for the general purposes of facilitating healing, and (2) into such special forms of treatment that may be required to combat distressing or urgent symptoms.

General Medicinal Treatment.—Carlsbad water is employed by many clinicians as a routine treatment, and is especially advocated by von Leube. A glass of Carlsbad water, either hot or lukewarm, is given in the fasting condition in the morning. If this quantity be insufficient to produce a daily movement, one or two extra doses may be given during the day, or the laxative effect of the morning dose may be increased by the addition of one-half dram to a dram of the powdered salts. If the natural Carlsbad water cannot be obtained, the desiccated salts prepared by Eisner and Mendelsohn may be employed—1 dram to 8 ounces of water being the dose ordinarily used. The object of the Carlsbad is to reduce hyperacidity, and it is therefore especially of service when hyperacidity or hypersecretion exist. It is of much less value in ulcers accompanied by normal or diminished acidity.

Silver nitrate is a drug that has been used for many years and may be employed in ulcers, either with normal or increased acidity, as an alternate to the carlsbad treatment. The usual way in which it is given and the one employed by the writer is to give three-sixths of a grain in distilled water, three times a day, one-half hour before eating, for three days. For the three successive days the dose is then increased to four-sixths of a grain, followed by a further increase to five-sixths of a grain for the third three-day period. This nine-day cycle is then repeated. It may happen that diarrhea will occur during this treatment. In the majority of instances this ceases spontaneously, but if it continues, especially if the patient is in a depleted condition, the medication should be either given in smaller doses or totally withdrawn.

A convenient prescription is:

R—Argenti nitrat.	gr. xvj
Aquæ destillatæ	℥ij
M. Sig.—5 minims equal gr. $\frac{1}{4}$.	

Dose—15 to 25 minims in distilled water, three times daily one-half hour before eating.

The silver treatment should not be continued for more than four weeks. At the end of a similar period of time it may be readministered.

Chronic indolent ulcers, especially those on an ambulant treatment, are often improved by lavage with 1 to 3000 solution of silver nitrate. Under this form of treatment the acidity is frequently reduced, and the subjective discomfort and pain are lessened. It is, however, doubtful whether there is any direct result on the healing of the ulcer. It may be necessary to abandon the treatment should diarrhea ensue.

Alkalies are imperative if hypersecretion be present. Among the antacids may be mentioned sodium bicarbonate, magnesia usta, magnesia carbonate, bismuth subcarbonate, and alkaline waters, of which Celestins Vichy is the type. Alkaline powders composed of antacids may be combined in a variety of ways. Bismuth subcarbonate is of use both as an antacid and as a mechanical protection to the floor of the ulcer. It is given in suspension in water, in doses proportionate to the acidity.

Fleiner recommends the bismuth treatment, should pain or distress occur when the diet is changed from fluid to semisolid, or from semisolid to solid, or should ulcer symptoms return after a cure. Given during an ulcer cure, the bismuth treatment is often of service; but given without other adjuvants of treatment, such as a rigid diet and bodily rest, it can only serve to control subjective symptoms of pain and discomfort that may be due to hyperacidity, and can have no direct result upon the healing of the ulcer.

Under no circumstances should bismuth subnitrate be used, as the sharp crystals of this preparation may mechanically irritate the floor of the ulcer.

Oil, either pure or in emulsion, is of value in cases of ulcer with hyperacidity and spasm of the pylorus. The pure olive oil may be used, or in more sensitive patients, emulsion of sweet almond oil may be employed. The oil treatment is given less in active ulcer cure than in the cases of ulcer that are up and about and eating solid food. The ordinary dose of olive oil is a teaspoonful half an hour before each meal.

Atropine is of service to control pain, to reduce acidity, and to relax muscular spasm. It is, therefore, chiefly employed during attacks of acute hypersecretion from pyloric spasm.

Treatment of Special Symptoms.—Hemorrhage.—The drug of greatest service is adrenalin in 10-minim doses of 1 to 1000 solution. Such a dose may be given every quarter to every half-hour, diluted in a small quantity of water. Similar results follow the use of the desiccated extract in 2 to 10-grain doses. A reaction dilatation may follow the vasomotor constriction which adrenalin produces locally, but by this time a thrombus usually becomes firmly fixed at the bleeding-point.

Gelatin may be given either as a rectal injection or by mouth, a 3 per cent. solution being usually employed; given by rectum one or two pints may be employed. A dose by mouth is from half an ounce to two ounces, as hot as can be borne, is given every half-hour or an hour, provided that administration is not followed by vomiting. The writer's results with gelatin have been unsatisfactory.

Ergotine hypodermically is not as much used as formerly.

Calcium chloride in 20-grain doses may be given by rectum, and is especially advised by Boas. It seems of use only in repeated hemorrhages.

All attempts at vomiting must be discouraged so as to prevent the dislodgement of the thrombus that is nature's method of relief. Morphine may be given hypodermically to quiet nervous apprehension and restlessness, provided the past experience of the patient is that opiates do not cause nausea and vomiting. Apprehensive relatives and fussy attendants should be dismissed from the sick room.

In extreme cases in which hemorrhage continues in spite of all methods employed, lavage with iced water has been recommended by as high an authority as Ewald. Before such a lavage, the patient should receive a small dose of morphine hypodermically and the throat should be sprayed with a weak solution of cocaine, so as to reduce to a minimum any attempt at vomiting. The washing should be continued until it comes almost entirely clear. Any increase of hemorrhage during this manipulation has not occurred in Ewald's experience. An ulcer that is not about to perforate, will probably not be affected by gentle lavage, and the procedure is perfectly justifiable in extreme cases of hemorrhage after other measures have proved useless.

Bourjet introduces into the stomach 100 c.c. of a 1 per cent. solution of ferric chloride, which is immediately allowed to siphon out of the stomach again, and this procedure he repeats five or six times and claims good results from its use. Should hemorrhage be sufficient to cause extreme degrees of anemia, liquids must be introduced into the system either by hypodermoclysis, or in less urgent cases by saline irrigations or the Murphy drip. In severe cases direct transfusion should be resorted to.

Vomiting.—Vomiting is usually relieved by regulation of diet, oxalate of cerium, small doses of Fowler's solution, minim doses of carbolic acid, to which may be added 1 to 2 minims of dilute hydrocyanic acid. Menthol in half-grain doses and crème de menthe frappé may be used, but are not of any service in the acid vomiting of hypersecretion. Rebellious cases may require total abstinence. The vomiting of acid fluid, indicating hypersecretion, usually from pyloric spasm, is treated by alkalis in large doses, and atropine pushed to mild physiological limits. If the vomiting does not yield to this treatment gentle

lavage with alkaline solutions is far safer than to risk hemorrhage from the strain of repeated emesis. Abstinence from food is indicated during acute hypersecretion.

Pain.—Pain is regularly relieved during the first week of an ulcer cure if the ulcer be without complications. Heat is distinctly calnative to pain, while alkalis relieve by reducing the acidity. Pain from adhesions may be alleviated by rest and hot applications and by giving food in small quantities at a time. If pain should continue after the tenth day of an active ulcer treatment we are confronted either with complications, or the case is one of mistaken diagnosis—to this rule there are but few exceptions.

Perforation is a purely surgical complication, and immediate resource should be had to laparotomy. There are undoubtedly cases of spontaneous cure, but these should be regarded as medical curiosities and should not allow us to waste valuable time by an ineffective medical treatment.

Individual Methods of Treatment.—Each writer of experience has his own method of treatment which to him has yielded the best results. While the general rules of treatment are applied in all these individual methods, the details of management vary somewhat, and it seems wise to describe two methods most popular at the present time, and to add the method of treatment preferred by the author and commonly employed by him.

von Leube's Treatment.—The patient remains in bed the first ten days. On the eleventh day he is allowed to be up and about, although lying down for one or two hours after each meal is insisted upon for a number of weeks afterward. von Leube claims never to have seen bad results by letting the patient leave his bed as early as this, either in the return of pain or in the retardation of the healing process. During this time poultice applications as hot as can be given are applied every ten or fifteen minutes during the day, while the Priesnitz application is applied at night. A preliminary sterilization of the skin is advised to prevent infection from any possible blisters that may be caused by the extreme heat.

Should blisters occur, the skin should be washed with ether and followed by an application of dermatol. These hot applications are to be used only in cases without visible hemorrhage. Should occult bleeding be present, extreme heat is undesirable and the Priesnitz application should be employed.

In all cases of recent hemorrhage, local applications of ice-bags to the epigastrium are to be employed until the stools show that the bleeding has ceased. When this occurs the ice is discontinued and moderate heat by the Priesnitz method is employed. Extreme heat is not to be employed for at least three months after acute hemorrhage.

A glass of lukewarm Carlsbad water is given every morning in the fasting state. von Leube believes in its healing qualities in cases of ulcer.

Few drugs are used except bismuth or sodium bicarbonate. Alkaline waters may be taken. Constipation is relieved by an enema of Carlsbad salts, or, after the eleventh day, by a teaspoonful of a powder composed of pulv. rhei 20 parts, sodium sulphate 15 parts, sodium bicarbonate 7.5 parts. The diet during this treatment is as follows:

Patients with recent hemorrhages are treated by nutritive enemas for the first three days, after which they begin the diet upon which patients without hemorrhages are placed at once.

For the first ten days small quantities of boiled milk, meat extract, soup, and unsweetened biscuits are given. The fourth day the patient receives $2\frac{1}{2}$ pints of milk, 6 ounces of rusk or zwiebach, and some meat extract. In the next seven days gelatinous soups, rice, sago boiled with milk, raw and soft-boiled eggs, boiled calves' brains, and broiled chicken and pigeon without fat or skin are added. In the next five days the diet is increased by rare steak finely minced, potatoes, puree rice soups, and weak tea and coffee.

During the third and fourth week there are given tender beef, roast chicken and pigeon and squab, well-cooked venison or partridge, macaroni, and the soft part of white bread. From the fifth week onward a return is gradually made to ordinary food.

Lenhartz Treatment.—The Lenhartz treatment is based on the belief that the weakening of the patient by too strict enforcement of a rigid and insufficient diet so undermines recuperative processes that the proper healing of the ulcer is prevented. Small repeated feedings are begun with impunity even after recent hemorrhage, and there is given a concentrated albuminous diet which converts free hydrochloric acid into the loosely combined form, and prevents further erosion and irritation of the ulcerated area.

The requisite course of treatment extends over two weeks. Absolute rest in bed is insisted upon. Local applications of ice-bags are employed during the first ten days. He recommends the use of bismuth subcarbonate in 30-grain doses three times a day. Chronic ulcers with pronounced pain are treated by silver nitrate and by a limitation of liquids. Bland preparations of iron are given if anemia is present. The following articles of diet are given:

Fresh milk, iced; both milk and eggs placed in a glass tumbler, surrounded with cracked ice, and kept at the bedside—even the feeding spoon is kept iced at the same time. The eggs and milk may be given alternately in hourly doses, or may be mixed and given together. Granulated sugar is added to the eggs after the third day. Raw scraped beef; boiled rice and zweibach are given later. According to the follow-

ing schedule, cooked chicken finely chopped, chopped ham or beef are added with butter and given in large doses. After the tenth day broiled chop or steak may be substituted for the scraped meat; toasted bread may replace the zwiebach, and fine cereals may take the place of the rice. During the first ten days rigid adherence to routine is insisted upon, both as regards the quantity of each article of diet given at each feeding and to the totals of each article for the twenty-four hours. The food is given in hourly intervals from 7 A.M. to 9 P.M., and a complete rest of ten hours is allowed during the night. Beef broths are contra-indicated, owing to the extractives and spices which tend to induce hyperacidity. Lenhartz begins his diet in a few hours even after severe and repeated hemorrhages, and claims to have no disastrous results from this early feeding. The details of his diet are given in the following table:

Day.	Eggs.	Milk.	Sugar.	Scraped Beef.
I	2 drams per dose; total, 2 eggs.	4 drams each dose; total, 6 ounces.		
II	3 drams per dose; total, 3 eggs.	6 drams per dose; total, 10 ounces.		
III	$\frac{1}{2}$ ounce per dose; total, 4 eggs.	1 ounce per dose; total, 13 ounces.	20 grams added to eggs.	
IV	5 drams per dose; total, 5 eggs.	$1\frac{1}{2}$ ounces per dose; total, 1 pint.	20 grams added to eggs.	
V	6 drams per dose; total, 6 eggs.	14 drams per dose; total, 19 ounces.	30 grams.	
VI	7 drams per dose; total, 7 eggs.	2 ounces per dose; total, 22 ounces.	40 grams.	36 grams in 3 doses.
VII	4 drams per dose; total, 4 eggs. Also 1 soft-boiled egg every four hours; total, 4 eggs.	2 ounces per dose; total, 25 ounces.	40 grams.	70 grams with boiled rice, 100 grams in 3 doses.
VIII	4 drams per dose; total, 4 eggs. Also, 1 soft-boiled egg every four hours; total, 4 eggs.	$2\frac{1}{2}$ ounces per dose; total, 28 ounces.	40 grams.	70 grams with boiled rice, 100 grams in 3 doses.
IX	4 drams per dose; total, 4 eggs. Also, 1 soft-boiled egg every four hours; total, 4 eggs.	3 ounces per dose; total, 1 quart.	Beef same; rice 200 grams. Zwiebach, 40 grams in 2 portions.
X	4 drams per dose; total, 4 eggs. Also, 1 soft-boiled egg every four hours; total, 4 eggs.	Add cooked chopped chicken, 50 grams, also butter, 20 grams.	40 grams.	Beef same; rice 200 grams. Zwiebach, 40 grams in 2 portions.

XI to XIV. Interval of feeding made two hours, milk given in 6-ounce doses, with $\frac{1}{2}$ ounce of raw egg. Butter increased to 40 grams and various additions made, as detailed above.

By this treatment Lenhartz claims a mortality of from 2 to 3 per cent., and states that the recurrences of hemorrhage are less frequent than in other forms of treatment.

Spriggs,¹ however, has followed 21 cases treated by the Lenhartz method; 9 had a definite return of symptoms of ulcer, and 3 complained of constant indigestion.

Of the two forms of treatment, von Leube's and Lenhartz', the writer believes that better results are obtained by the former than by the latter in the average run of ulcer cases; but that when patients are debilitated by hemorrhage, by insufficient nourishment, or by prolonged or recurrent vomiting, the abstinent treatment and insufficient diet during the first week or ten days of von Leube's treatment tend to lower the vitality of the patient to such a point that recuperative processes are held in abeyance. It is in this class of cases that the Lenhartz treatment is particularly advantageous.

Writer's Method of Treatment.—The patient is kept in bed for four weeks. During the first ten days he is not allowed to arise even for toilet purposes. This enforcement of body rest is an essential of the treatment.

Treatment for the First Three Days.—Absolute abstinence is enjoined for the first seventy-two hours in all cases, whether or not hemorrhage or occult bleeding be present. The patient eats nothing after his dinner the night before beginning treatment, so that the last twelve hours of starvation are passed in the day rather than in the night.

During these three days mouth washes are used frequently, to minimize mouth sepsis and to assuage the feeling of thirst. A cleansing enema is given early in the first day. Nutritive enemas are not employed, nor is enteroclysis advised except in the case of those who are weakened by hemorrhages, insufficient nourishment, recurring vomiting, or who are constitutionally in a condition of lowered vitality. Decinormal solutions of sodium bicarbonate are preferable to the saline solutions usually recommended for the reasons previously given, and the fluid is best introduced by some one of the modifications of the Murphy drip. In milder cases in which the only indication is to relieve thirst, retention enemas of the soda solution may be given.

Drugs during this period are but rarely used, exceptions being made in the case of recent hematemesis or occult bleeding, and in cases of continuous secretion with or without acid vomiting.

During the entire period of treatment hot moist applications are to be employed, except when visible hemorrhage has occurred at any time within the previous three weeks, and the hotter they can be used

¹ British Medical Journal, April 3, 1909, p. 825.

the better. The writer has never used such extreme degrees of heat as von Leube, who applies hot poultices every ten to fifteen minutes throughout the day, but who continues this treatment for only ten days. The writer recommends only such heat that can be used without excessive discomfort, using the electric pad by preference day and night, or when this cannot be employed, the Priesnitz application, changed every hour during the day, twice at night in the first two weeks, and once at night in the latter half of the treatment. The external application must be continued at least one month.

In cases of visible and evident hemorrhage, ice-bags are to be applied constantly until all traces of blood have disappeared from the stools, and are to be then succeeded, not by extreme heat, but by the Priesnitz applications, not hot, but warm and reapplied only every six hours.

Treatment from the Fourth to the Seventh Day.—Fourth day: At the end of seventy-two hours feeding by mouth may be begun, by giving peptonized milk in 2-ounce doses, and a similar quantity of Celestins Vichy or of a solution of sodium bicarbonate, gr. v to 2 oz. water on the intervening hours, so that the patient receives 2 ounces of liquid every hour. Sleep must not be interfered with. On the fifth day these quantities are increased to 3 ounces, on the sixth day to 5 ounces, on the seventh day the milk is increased to 7 or 8 ounces, while the alkaline water, given at the same stated periods, is reduced in quantity to suit the desire of the patient.

The author's method of peptonization to be recommended:

To 1 pint of milk is to be added $\frac{1}{4}$ pint of water, and the mixture is to be divided into two equal parts. Boil one part, and immediately afterward add the other. Stir in the contents of one of Fairchild's peptonizing tubes, and set the bottle in warm water for one and one-quarter hours. Bring rapidly to a boil and keep on ice. The completely peptonized milk should have a slightly bitter but not unpleasant taste.

No drugs are usually employed during this period unless indicated to meet special conditions, such as acidity, vomiting, or hemorrhage. External applications are continued. The bowels are moved daily by single enemas.

Should the patient be one who is habitually constipated, Carlsbad treatment may be begun.

Treatment during the Second Week.—The diet may now be enlarged by the substitution of the following articles for any one of the doses of milk: Junket, arrowroot gruel, milk toast, creamed macaroni, malted milk, blanc mange, farina, and hominy or cream of wheat with cream and sugar. Not more than 5 ounces of any one of these should be given at any one time, and the system of two-hour feedings continued. Only one article is given at a time. Celestins Vichy or the soda solu-

tion may be taken as often as desired, but not in greater quantity than 4 ounces at any one time. External applications are to be continued.

Two methods of medication may be employed: the Carlsbad treatment and that by silver nitrate. The details of both forms of treatment have been previously described (see p. 176). Between these two forms of medication the writer sees very little to choose, results seeming to about the same with one as with the other. The Carlsbad treatment is generally to be given the preference to those who are habitually constipated and flatulent, with coated tongue and other evidences of hepatic insufficiency. The silver nitrate cycle seems to be indicated especially in these ulcers, with clean tongues and regular bowel functions, which are accompanied by a heightened acidity, and usually with persisting pain.

Treatment during Third Week.—During the third week the only change is in the enlargement of the diet, there being gradually added mashed potatoes, purées of any kind *not* made with meat stock, creamed or boiled fresh fish, soft boiled or poached eggs, the soft part of pumpkin pie, custard, and mashed vegetables that can be put through a purée sieve. Soft bread well masticated or crackers are allowed. Several articles of diet may be given at a time, and the feeding interval may be lengthened to every three hours. During the third week milk is usually discontinued.

Treatment during Fourth Week.—During the fourth week the patient is allowed to sit up a portion of each day, and the external applications are gradually diminished. The only other change in the treatment is the addition of creamed chicken, tender squab, lean boiled ham, and minced veal. Should anemia be present, iron in some bland form may be given. Given earlier than this in the treatment it is not generally well borne. Strychnine, nux vomica, or eserine are frequently valuable adjuncts.

After-treatment.—The diet of the fourth week is to be continued for at least a month before resumption of a more varied menu, the quantity given at any one time may be gradually enlarged, so that the patient is allowed three larger meals and two smaller meals a day. Eating at night is not recommended. For at least six months red meat, scratchy articles of food, raw fruit, and fruit juices, ice cream, ice water, and all highly seasoned and spicy articles of food must be forbidden. A little whisky and water at dinner may be allowed, but cocktails, champagne, and the heavier wines must be prohibited. Smoking should be in moderation and only after eating. Tea is unadvisable; weak coffee, especially caffeine-free coffee, such as the "Dekafa" of Merck & Co., largely diluted with milk is allowed at breakfast only.

The author's convalescent ulcer diet is here given:

Breakfast. Fine cereal, with cream and sugar, such as farina, vitos, hominy, toasted corn flakes. Coarse cereals, such as oatmeal and cracked wheat, not allowed. No dry toast, bread crusts, or hot bread. May have soft parts of bread, milk, or cream toast, or crackers thoroughly masticated. No tea or coffee. May have cocoa with cream and sugar; milk or malted milk. Two soft-boiled or poached eggs.

11 A.M. Choice of malted milk, junket, cup custard; top milk or cream, or milk and cream. Egg shake without wine or brandy. Russell's emulsion. Two raw eggs. Puree of any kind, made without meat stock.

Luncheon: Puree of any kind made without meat stock. Oysters in any form; if raw, to be taken with only a little lemon juice. Creamed or milk toast. Creamed or boiled fresh fish. Mashed or baked potatoes, without pepper. Spaghetti or macaroni. Creamed or minced chicken. Butter should be taken freely, preferably unsalted. Farinaceous dessert, such as farina, tapioca, corn-starch, blanc mange, rice pudding, custard. No ice-cream, fruit ices, cakes, or fruit of any kind.

4 to 5 P.M. Same variety as at 11 A.M., but may have additional choice of cocoa with cream and sugar, or a farinaceous dessert. No tea allowed.

Dinner. Same variety as at luncheon.

10 P.M. Same variety as at 4 P.M.

Water to be scanty at meals, cool but never iced. Celestins or Saratoga Vichy preferably to pure water.

No wines or alcohol in any form.

Smoking allowed only in the greatest moderation, and never between meals.

In those cases in which the silver cycles were administered this drug should be discontinued after the fourth week and the Carlsbad treatment substituted. In those treated primarily by the Carlsbad salts, this should be continued. It is more convenient and equally efficacious to reduce the dosage to one hot glassful one-half hour before breakfast.

Treatment of Hemorrhage.—Single large hemorrhages are best treated medically. Operative interference either is not necessary or the patient is so exsanguinated as to render an operation of such magnitude unjustifiable. Abstinence from all food or drink should be absolute. Ice-bags are to be applied to the epigastrium to promote firm contraction of the stomach. The general methods of combating syncope and other symptoms of acute profound anemia are to be resorted to, such as lowering of the head, elevation of the foot of the bed, and the giving of liquids subcutaneously or by the bowel.

Morphine is to be given only when there are frequent attempts at

vomiting or whenever restlessness and apprehension prevent the mental and body quietude that are necessary to promote the formation of a clot at the bleeding point.

Adrenalin is the one drug which seems serviceable. The solution of adrenalin chloride (1 to 1000) may be given in 10-minim doses every hour. The reaction dilatation of the bloodvessels that follows its use need not be considered in acute hemorrhages.

Gelatin has not seemed to be of service. It may, however, be employed in 3 per cent. solution by rectum. Given in this way it can do no harm, possibly a little good, while the effect of introducing liquids into the system by use of such a solution is often useful in moderating the severity of the symptoms of exsanguination. Gelatin by mouth is not to be recommended, as it violates the principles that nothing should be allowed to enter the stomach during the time of active hemorrhage.

Should the hemorrhage continue in spite of the above treatment, and the patient be accustomed to the tube, gentle lavage with ice water may be used and continued until the return flow is nearly clean. If the patient be left in an exhausted condition the Lenhartz diet may be given as soon as bleeding ceases.

Chronic hemorrhages are to be treated by gelatin by mouth in doses of a 3 per cent. solution every hour or so, by calcium chloride in 20-grain doses by rectum every four hours. Adrenalin is not to be employed for any length of time, owing to the vasomotor dilatation that may follow its prolonged use. The continuance of repeated hemorrhages during a liquid diet may render operation advisable.

The subcutaneous injection of blood serum which has given such striking results in hemorrhagic diseases of the newborn, has apparently been of great benefit in the control of hemorrhage from gastric and intestinal ulceration. Human serum whose suitability has been previously tested, as for transfusion, is best, although when such tests cannot be made, as in emergencies, untested serum may be used, as the risk involved from hemolysis and agglutination is probably very slight. Next best in order of safety are rabbit and horse serum. When they are used the danger of anaphylaxis, though somewhat remote, must be borne in mind:

From 10 to 15 c.c. of serum are injected at one time, and this injection may be repeated on successive days, if necessary, or even at shorter intervals. The blood is obtained under the strictest aseptic precautions from a superficial vein, and collected in sterile flasks which are kept on ice until the serum has sufficiently separated.¹ This

¹ Welch's technique may be found in *Amer. Jour. Med. Sci.*, cxxxix, 213.

is best done by one who has had experience in serology. The effect of the serum treatment probably begins about six or eight hours after the injection, and lasts for several days.

In urgent cases of hemorrhage direct transfusion of blood is indicated.

Occult Bleeding.—The stools should be tested for the presence of occult blood at least every three days during the first fortnight. The presence of blood traces during this period indicates that the ulcer is not properly healing, and that no further increase in the diet is permissible, provided that the meagreness of the diet is not carried to such an excess as to reduce to too low an ebb the vitality of the patient, and thus to retard the healing process. Every radical change in diet—the substitution of semisolid for liquid food, or the giving of solids instead of soft or semisolid nourishment, is to be continued only when such a change is not followed by a positive return of the blood test. Should the occult bleeding persist in spite of a diet that is inadequate to maintain a proper preservation of bodily flesh and strength, the patient should at once be placed upon the Lenhart treatment, which should be continued no matter what may be the reaction of the blood test.

If during the third and fourth week bleeding continue, suspicion should always be entertained of beginning malignancy. Record of blood counts and of body weight must be systematically reported. Loss of weight or progressive diminution in the percentage of hemoglobin during the second month of treatment, with or without positive blood reactions, afford sufficient reason for advocating surgical exploration.

Treatment of Hypersecretion.—If chronic hypersecretion is present it is rarely sufficiently pronounced to demand any special treatment during the first three days, and, moreover, it usually subsides entirely after the giving of food is discontinued. Should the patient complain of hyperacidity and heart-burn, small doses of milk of magnesia may be given, or bismuth subcarbonate in suspension, giving only such quantities as may be necessary to relieve the burning.

Acute Hypersecretion.—The treatment of acute hypersecretion, with burning and distress, and the vomiting of acid watery fluid is the same whether it occurs during the first three days or later in the course of treatment. Rest to the stomach and doses of alkalies sufficient to neutralize the acid are the essentials of the treatment. Sodium bicarbonate may be an ingredient of any of the combinations of alkalies administered, but should only be given in small quantities, as the resulting liberation of carbon dioxide may cause the overdistention of the stomach, which we are anxious to avoid.

Gentle lavage with weak alkaline solutions may be employed in cases of excessive vomiting, especially if the patient be accustomed to the use of the tube, and at the close of the procedure an ounce of a 3 per cent. solution of anesthesin in olive oil may be introduced. Unless idiosyncrasy exist, atropine should always be given subcutaneously in doses sufficient to cause mild physiological effects, even though annoying dryness of the tongue be increased by its use. It is only when the severity of the attack becomes modified that the drug may be given by mouth, either as the alkaloid itself or in the form of tincture of belladonna. When hypersecretion ceases, the drug is to be withdrawn—its use as a routine procedure as recommended by some writers is not to be advised.

Einhorn has devised a method of feeding which he terms duodenal alimentation. His apparatus consists of a perforated aluminum capsule to which is attached a thin, soft, India-rubber tube having three markings: the first indicating the distance from the dental arcade to the cardia; the second, from the dental arcade to the pylorus, and the third mark indicating the distance from the dental arcade well into the duodenum.

The capsule is to be swallowed at night, the end of the tube being attached by a piece of plaster to the cheek. The following morning it is to be hoped that the capsule is in the duodenum; but this must be demonstrated as a fact before beginning the feeding. The indications that the capsule is in the duodenum are, (1) that the tube has passed to the third mark; (2) gentle traction on the tube develops a sense of greater resistance than if the capsule were lying free in the stomach; (3) aspiration removes a golden-yellow duodenal juice; and (4) water given by mouth cannot be aspirated through the tube.

The food injected consists of milk, sugar of milk, and raw eggs, in the proportion of one glass of milk, one egg, and a tablespoonful of sugar of milk. The amount at first injection is 100 c.c. every two hours, from 7 A.M. to 9 P.M., increasing gradually so that 280 to 300 c.c. are given at each feeding, representing approximately 2800 calories. The food mixture should be gradually heated so as to avoid lumpiness from the coagulation of the egg, and then strained. The food should be given at body temperature, and should enter the duodenum slowly. Failure in either particular will cause flatulence and distress.

Einhorn uses a glass syringe for injection. William Gerry Morgan uses a glass irrigating jar, setting the petcock so that the nourishment flows in twenty-five minutes. At the termination of each feeding a syringeful of water should be injected at body temperature, the petcock closed, the syringe filled with air, the petcock opened and air injected, after which the petcock should be closed and the syringe

disconnected. This procedure keeps the tube clean and clear. These details should never be neglected.

It is claimed by those who have used this method, that nourishment can be given sufficient to maintain body strength and weight, and that the freedom from irritation and gastric secretion allow of the ready healing of ulcer. The writer's experience with duodenal alimentation has been quite limited, as he has found that the majority of patients do not readily accustom themselves to the constant presence of the tube, and that flatulence and distress are frequently occasioned, in spite of every care in the preparation and injection of the nourishment. Whether the end-results of the duodenal alimentation are superior to those of the other forms of treatment cannot be decided at present, as not sufficient time has elapsed for the final end-results to be tabulated.

Indications for Surgical Treatment.—During the past few years, the relative indications for medical and surgical treatment of ulcer have been fully and freely discussed, with the result that at the present time physicians and surgeons have come to think and act in perfect harmony with each other. There is no such thing as an exclusive medical treatment, nor can it be affirmed that ulcer is a purely surgical disease—but, on the other hand, while some ulcers are to be treated medically, and others with their complications call imperatively for surgical treatment, the majority are grouped near the borderline. In these doubtful cases the main indication for surgery is the failure of a previous medical treatment to obtain beneficial or lasting results.

Acute Uncomplicated Ulcers.—Acute uncomplicated ulcers are best treated medically. This is conceded by all surgeons who often designate this form as “medical ulcer.” Relief by medical means is usually prompt and lasting.

Chronic Uncomplicated Ulcers.—Chronic uncomplicated ulcer should not be regarded as surgical until after a rigid and systematic course of medical treatment, the symptoms persist or recur. It must be emphasized that the treatment should be thorough, and continued for a sufficient length of time, as medical failures are due more often to half-hearted and insufficient treatment than to actual limitations of the healing art. In general terms it may be said that ulcers that do not yield to two months' active treatment will prove resistant except to surgical procedures. Some consideration must, however, be paid to outside conditions, such as the amount of time that can be given to treatment, or the probability of the patient taking sufficient care of himself during the convalescent period, to prevent relapse or recurrences. In the case of a laboring man whose family is dependent upon his daily wage for the necessities of existence, and who is apt

on leaving the hospital to disregard all dietetic rules and conventions, it may be better to recommend surgical intervention earlier than would be advisable in the case of those whose intelligence and environment allow of a conscientious observance of the details of treatment.

If after a vigorous medical treatment the symptoms persist, or if in spite of every precaution there should be relapses, indicating that the healing of the ulcer has not been complete, surgical treatment is indicated. The mortality rate and the percentage of cures are both on the side of surgery in these cases.

Exploration is demanded without loss of time in all cases in which there is a suspicion of malignancy.

Single Large Hemorrhages.—Single large hemorrhages are best treated medically. If the bleeding ceases by medical means no harm is done, while the operation of opening the stomach and ligating the bleeding point, when perforated during a period of acute anemia, is extremely hazardous. Hemoglobin tests under 35 per cent. contra-indicate operation except in emergencies in which great risks may be taken. On the other hand, hematemesis that ceases spontaneously is often accompanied by anemia even greater than this.

Recurring Hemorrhages.—Recurring hemorrhages, if not relieved by a course of medical treatment, may properly be placed on the surgical list, especially if the continued loss of blood occasion a progressive anemia. In these cases operation should not be too long deferred, as there may be complicating malignancy present. Cases in which occult bleeding follows every attempt to place the patient upon solid food, after a regular ulcer treatment, should invite surgical intervention.

Perforation.—Perforation, either acute, subacute, or chronic, is a purely surgical complication. Acute perforations demand instant operation, as every hour that elapses until the aperture is closed diminishes the patient's chance of recovery. Immediate laparotomy is called for alike in those cases with but slight amount of initial shock, such as are often encountered with duodenal perforations, and those in whom initial shock is profound. It would be a fatal error in judgment to temporize in the one case until the indications for interference become more manifest, or to defer laparotomy on the other in the hope of bringing the patient into a better physical condition to withstand an operation.

Obstruction and Adhesions.—Pyloric obstruction, hour-glass contractions of the stomach, or persistent adhesions which interfere with the proper drainage of the stomach into the bowel are to be treated surgically, although in the majority of instances preliminary treatment by lavage and diet may so improve the general condition that the operation is performed with a minimum risk.

Adhesions whose presence is only to be surmised and which do not interfere with the proper motility of the stomach are best left untreated. An exception may be made in those perigastric adhesions, especially those usually between the lesser curvature of the stomach and the under surface of the liver, which are dragged upon by physical exertions, or even during locomotion, and give rise to such discomfort as to render it impossible for the patient to be up and about doing his work.

CHAPTER V

EROSIONS AND RARE ULCERS

HEMORRHAGIC EROSIONS

THE term hemorrhagic erosions indicates those minute ulcers of the stomach, single or multiple, which invade only the more superficial portion of the gastric mucous membrane and which heal completely, leaving no trace of any loss of continuity whatever. Pathologically they are ulcers, but it is convenient to describe them under a separate heading as their clinical course is somewhat different from the deeper erosions ordinarily described as gastric or duodenal ulcers.

It is a disputed question whether erosions and ulcers represent two distinct ulcerative processes or whether an ulcer is not merely a well-developed and more extensive erosion. Those who affirm that the erosion is merely a small shallow ulcer which may extend in depth and persist as the classical ulcer, base their conclusions on the finding at post mortem of multiple superficial erosions and well-developed ulcerations in adjacent portions of the same stomach. This is the view held by Gerbardt,¹ Nauwerk,² and Dieulafoy, and in this opinion the writer is inclined to coincide and to believe that the subdivision of ulcer from erosion is useful for purely clinical reasons rather than because there is any essential difference in the pathology of these two kindred conditions.

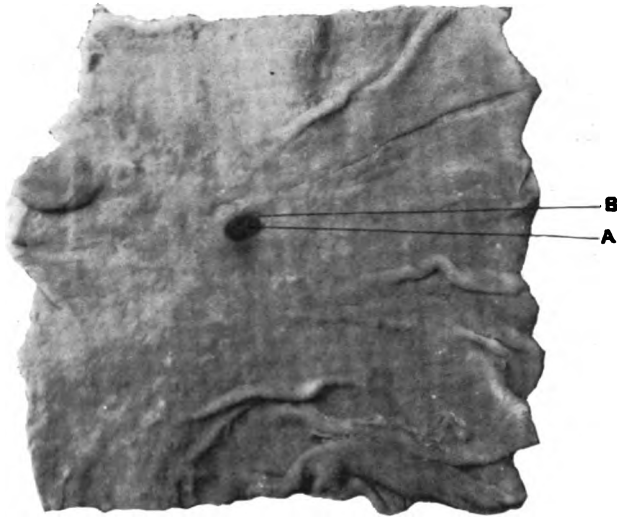
Whatever way, however, this question is answered, one fact remains undisputed, namely, the passage from a shallow erosion to the typical deeper ulcer is one of great rarity. Erosions may appear both in an acute and in a chronic form.

Acute Hemorrhagic Erosions.—The formation of hemorrhagic erosions as the name implies, consists of two stages. In the first stage there occur large or small multiple hemorrhages into the substance of the mucous membrane, appearing as purplish brownish or black patches, which later become digested, leaving superficial ulcers. Such resulting erosions may be solitary or multiple. The size varies from minute pore-like excavations to broad shallow depressions, as if the surface

¹ Virch. Arch., cxxvii, 85.

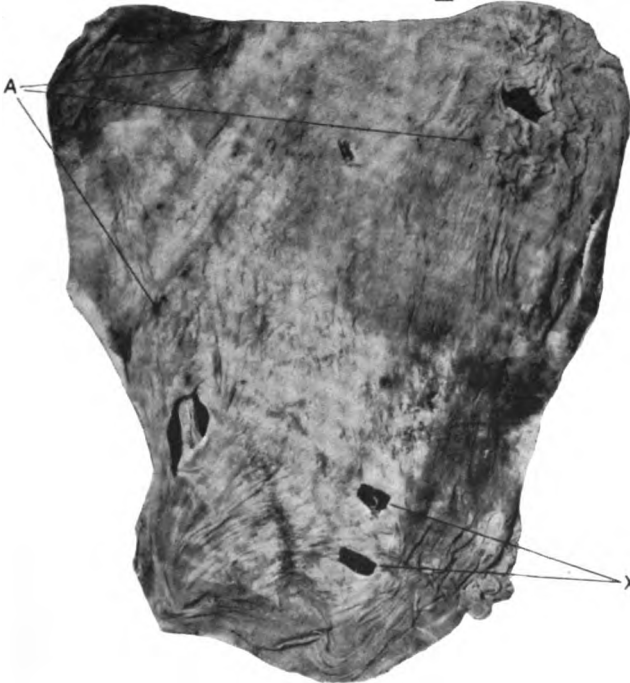
² Münch. med. Woch., 1895, p. 38 and 39; 1897, p. 35 and 36.

FIG. 32



Hemorrhagic erosion of stomach. The area of hemorrhage is well seen at A; the superficial loss of substance at B. (From the Pathological Museum of the Mt. Sinai Hospital, New York.)

FIG. 33



Multiple hemorrhagic erosions of stomach. Many of these hemorrhagic areas can be seen as at A, A, A. Several pieces of the stomach, as at X, X, have been removed for microscopical examination. (From the Pathological Museum, Mt. Sinai Hospital, New York.)

of the membrane had been lightly brushed off with the fingers. They may be extremely difficult to find at post mortem or in the living stomach at operation, and may even totally elude the most careful search. Postmortem digestion rapidly effaces these very superficial ulcerations. Although they do not invade the muscularis, erosions of small arteries may occur, which may result in serious or even fatal hemorrhage. As distinguished from the ordinary forms of ulcer, they tend to heal rapidly and completely, leaving no trace behind as evidence of their existence.

In many cases similar erosions are found in the duodenum and jejunum, and there are often subperitoneal hemorrhages.

Etiology.—Gastric erosions may form as the result of any one of three distinct conditions.

Toxic.—The fact seems well established that in many septic conditions bacteria may either invade the membrane of the stomach and cause small areas of necroses, or toxins, circulating in the blood may act as endothelial poisons on the wall of the arterioles and thus allow the escape of blood into the tissues. A very good illustration of the type of direct bacterial invasion is given us by Dieulafoy in his description of pneumococcal gastritis with ulceration.

His patient was admitted to the hospital with pneumonia; abdominal pain, tympanites, vomiting, and diarrhea were present. The day of the admission he vomited a pint and a half of blackish fluid-like coffee grounds and the stools contained blood. Death occurred on the following day.

Postmortem examination showed pneumococcic infection of the lung, pleura, pericardium, and peritoneum. There were erosions in the mucous membrane of the stomach, multiple and exceedingly minute, and in the interglandular stroma around the edges of these erosions were pure cultures of the pneumococcus.

A further proof that erosions may be due to direct deposition of bacterial organisms in the tissues is afforded by the fact that in many instances small miliary abscesses of the mucous membrane are found associated with the erosions. In a case of Giraudeau's, reported by Dieulafoy, the ulceration involved an arterial branch and in the neighborhood of the vessel masses of leukocytes were seen representing true miliary abscesses, with numberless microorganisms in chains.

It has been long surmised that toxins of many kinds may be the cause for multiple erosions of the stomach. This mode of origin seems quite definite in the case of uremic ulcerations and the duodenal ulcerations that complicate burns of the surface of the body. Although definite proof is lacking, it would seem that poisons formed from the pent-up secretions in appendicitis or gall-bladder infections were capable of

assuming the role of gastrotoxins and of producing definite gastric lesions. There is no doubt that gastric erosions occur frequently enough with infections of the appendix and gall-bladder, but whether due to the elaboration of endotoxins or to pylorospasm with attending traumatism of the mucous membrane of the pyloric antrum is not always a question of easy solution. Toxic erosions of the gastric mucosa is the usual cause for the gastric hemorrhages that accompany a splenic anemia and certain disorganized states of the blood.

Retrograde Embolism.—It is supposed that in some instances retrograde embolism from detached thrombi, may be the cause for the lesion, the thrombi being formed in the ligated veins of the mesentery or omentum.

This is the view held by von Eiselberg to explain postoperative hematemesis of the abdominal operations, but it seems doubtful if it will explain more than a very small minority of such cases.

Muscular Contractions and Pylorospasm.—It is possible that muscular contraction of the pyloric end of the stomach and pylorospasm induced by irritative lesions in the embryological midgut or its derivatives may inflict sufficient traumatism upon the mucous membrane of that portion of the stomach as to devitalize certain areas of it and allow of autodigestion. This mode of origin of erosion would explain the largest number of cases which occur as complications of gall-bladder or infections, and of both acute and chronic complications of the appendix. That such a muscular spasm does occur is well established, both by the *x*-ray and by the presence of visible contractions at the time of operations done for the relief of chronic appendicitis or the gall-bladder disease.

Symptoms.—Before the predominant symptoms of hemorrhage occur the patient may complain of more or less pain in the region of the stomach and the raising of acid fluid which may contain blood. A disagreeable taste in the mouth that is quite unlike that common to the ordinary type of indigestion may be a noticeable factor, and may seriously interfere with the appetite of the patient. When blood is finally vomited, its taste then appears to be identical with that complained of before the actual emesis. During this time the patient may have a peculiarly ill appearance, and is often of an ashy paleness. These symptoms occurring in a patient who has had an abdominal operation should excite suspicion, and examinations of the stools should at once be made for occult blood. These symptoms are apt to occur within three days after operation.

The most prominent symptom of erosion is hemorrhage, either a single large hemorrhage or smaller hemorrhages frequently repeated and associated with melena. In the majority of instances a profuse

and lightning-like hematemesis is the first indication of the illness, and may be so severe that death results from acute anemia during the first attack.

Subsequent hemorrhages are not infrequent. In those cases which have been operated on for the relief of hemorrhage or which have gone to post mortem it has often been well-nigh impossible to find the point or points from which the hemorrhage has taken place. In many instances the bleeding has occurred from numerous points, so that it has been said that the mucous membrane weeps blood. These obscure cases in hemorrhage have been termed by Hale White, "Gastrostaxis" or the bleeding from a stomach which shows no visible ulcer, but whether or not such cases really occur without ulceration or erosion may be doubted. It seems most probable to the writer that these are merely cases in which the bleeding-points are not found owing to their minuteness.

It has often happened that a cause for gastric oozing has not been found by the one observer, although minute, pore-like erosions accountable for the hemorrhage have been demonstrated by a subsequent examination of the same specimen.

Fenwick¹ says that in one instance he was able to demonstrate by artificial injection the source of a fatal hemorrhage that had eluded the most careful search at the necropsy.

Prognosis.—The prognosis is usually good, as the erosions ordinarily heal rapidly and are not followed by the symptoms of chronic ulceration. Even in cases in which anemia is extreme and threatening, recovery may be hoped for. In two cases reported by Dieulafoy the patient made a satisfactory recovery after a blood count of only 630,000. When, however, hemorrhages are associated with severe general infections, the prognosis is exceedingly grave.

Treatment.—The most clean-cut and definite point of the treatment is that the cases are to be treated on purely medical lines; with acute hemorrhages or erosions surgery has nothing whatever to do. The medical treatment is that of acute ulcer,

Conditions of extreme anemia may require hypodermoclysis or the infusion of salines by the Murphy drip. In urgent cases direct transfusion of blood as an emergency operation may be advisable. Adrenalin may or may not be of service. Calcium salts and solution of gelatin have been disappointing in their results. The hypodermic use of rabbit or horse serum has been recommended, and the writer has seen cases in which such a serum treatment has apparently been of use in controlling the loss of blood.

¹ *Lancet*, March 12, 1910.

Chronic Hemorrhagic Erosions.—This term is used to describe the condition in which bits of mucous membrane are found in the lavage water of the fasting state, usually associated with chronic gastritis or achylia, and characterized clinically by epigastric pain after meals. This condition has been fully described by Einhorn, Sansoni, and Quintard, and by these writers is considered to be a clinical entity. The characteristic features of the ailment appear to be the finding of small fragments of mucous membrane occurring quite constantly in the return flow of the stomach washings in the fasting state. Pain is a fairly constant symptom usually occurring soon after meals, attaining its height within a moderate time and then gradually disappearing as the stomach empties itself. The pain, according to some writers, may be exceedingly severe, according to others, quite insignificant, constituting a feeling of distress rather than a true pain. Bleeding may occur and is usually of the occult type. Sansoni has, however, noted well-marked hemorrhages in several of his patients. It is said that the diagnosis should be suspected whenever symptoms of ulcer, such as pain and hemorrhage, are complained by a patient who is suffering from chronic gastritis or achylia.

The writer believes that chronic hemorrhagic erosions thus described do not occur as a distinct clinical entity. It is a well-known fact that the mucous membrane in achylia is particularly friable, so that in taking the test breakfast we find pieces of mucous membrane, often of considerable size, in the eye of the tube. The evulsion of such pieces is not followed by any unpleasant results. A similar experience often occurs with the passage of the tube in chronic gastritis. The writer would therefore regard this form of hemorrhagic erosion as an artefact and one that becomes more and more rare the more carefully we select a tube whose apertures are soft and rounded. As to pain in achylia being due to hemorrhagic erosions, it would be interesting to know in how many of these patients the pain was due to gall-bladder or appendicular disease.

A history of one of the writer's patients was at one time considered by him typical of hemorrhagic erosions, the so-called "gastritis anacida ulcerosa" of Sansoni, until the patient was operated upon for chronic appendicitis, after which all pain and gastric discomfort disappeared.

Mrs. A. C., aged thirty years, was well until five years ago when without apparent cause she began from time to time to suffer from nausea and vomiting. Four years ago there was added a pain which started in the epigastrium and thence downward and to the back, coming shortly after eating and gradually wearing away. Nausea and vomiting might accompany the acme of pain. There would be intervals of time during which she was free from all symptoms.

Physical examination showed slight tenderness over McBurney's point, a moderate degree of gastroptosis was present, gall-bladder neither palpable or tender. Gastric analysis showed chronic mucous gastritis of the achylia type. On washing the stomach bits of mucous membrane, averaging the size of the head of a pin, with on one occasion a moderate amount of blood, were fairly constantly found. She was treated by lavage with 1000 to 3000 silver nitrate, and by an ulcer diet without much success until the appendix was removed, after which she was perfectly well without return of her stomach symptoms.

The writer does not wish to make the positive statement that this form of exfoliating erosions does not exist, but would regard the question of its clinical entity as not proved, or even probable at the present time.

JEJUNAL AND GASTROJEJUNAL ULCERS FOLLOWING GASTROJEJUNOSTOMY

Ulceration of the jejunum near the site of the anastomosis that is made in the operation of gastrojejunostomy has been considered one of the dangers that may follow such an operation.

According to Paterson¹ we are justified in estimating the probable risk of jejunal ulcer following gastrojejunostomy at a little under 2 per cent. It is, however, a question whether this form of ulcer is as common today, with improved methods of technique, as it was in the early days of gastric surgery. Nevertheless of 72 consecutive cases of gastrojejunostomy done for the relief of gastric or duodenal ulcer recently reported by Sherren,² jejunal ulcer complicated the convalescence in two instances. Both occurred after the posterior no-loop operation. von Roojen reports 3 cases of peptic jejunal ulcer in which no operation had previously been done.

Mayo³ writes that of 1141 gastrojejunostomies up to December 31, 1909, by himself and brother, C. H. Mayo, in not a single instance did true jejunal ulcer occur, nor had any such cases come to his clinic in which gastrojejunostomy had been performed by other surgeons. Mayo, however, makes a difference between ulcers that are implanted at the site of the anastomosis and the true jejunal ulcers that occur in the jejunum itself without involving the anastomotic ring.

Paterson estimates that in nearly one-third of the recorded cases the ulcer was probably gastric rather than jejunal, and in all prob-

¹ *Annals of Surgery*, August, 1909.

² *Lancet*, July 13, 1912, p. 76.

³ *Surgery, Gynecology, and Obstetrics*, March, 1910.

ability originated in the gastric mucous membrane surrounding the anastomotic opening. For ulcers at the site of the anastomosis he suggests the terms "gastrojejunal" to differentiate them from the ulcers which occur in the jejunal loops, the "jejunal ulcer" proper. The writer adopts this subdivision of postoperative ulcerations into these two groups.

Jejunal Ulcers.—It is curious that males are more affected than females with this disorder, either because men are more indiscreet in their diet than are women, especially in the use of alcohol after operations, or because, as Paterson suggests, more gastrojejunostomies are done in men than in women. Of 50 cases reported by Paterson in which the sex of the patient was mentioned, a proportion of 78 per cent. of men is recorded. Schostak¹ found 32 males were affected in a series of 35 cases. The complication has occurred after nearly all the variations of the operation, but seems to be less frequent with the modern short loop than in the older method in which a long loop of the jejunum was used for the anastomosis.

Pathology.—The ulcer may occur in the jejunum near the anastomosis, rarely at a point farther than 6 or 7 c.c. from the stoma. A single ulcer is the rule, although several ulcers have been found grouped together.

In some instances there is marked dilatation of the afferent loop, occasionally of both the afferent and efferent loop. In a case reported by Percy² both loops were enlarged to an external diameter of $2\frac{1}{2}$ inches. Their walls were stiff and thick, and without the collapsible feeling normal to the small intestine.

Such an ulcer may perforate with or without limiting adhesions, so that general peritonitis or a localized peritoneal abscess may ensue. Adhesions may form with the neighboring parts, especially the anterior abdominal wall and the transverse colon, and by extension of the necrotic process fistulas may form. The microscopical pathology is identical with that of gastric and duodenal ulcer.

Etiology.—The most potent cause in the formation of postoperative jejunal ulcer is the passage of hyperacid gastric juice into a part of the intestine which is not naturally resistant as is the duodenum to its erosive action. Katsenstein's experiments on autodigestion are interesting. He has proved that the introduction of the normal duodenum into a living stomach of the same animal is unattended by an erosive action on the loop so introduced, but that introduction of a loop of jejunum under the same conditions is followed by its total erosion.

¹ Beiträge z. klin. Chir., 1907, lvi, 360.

² Jour. Amer. Med. Assoc., April 9, 1910.

His argument is that those tissues which produce, or are normally bathed in gastric juice, are ordinarily immune from its eroding effect, but that all other tissues succumb to the digesting power of this fluid when immersed in it in the living state.

Hyperacidity was present in 13 out of the 18 cases recorded by Paterson in which the gastric analysis were made. Hyperchlorhydria occurred in 17 out of 21 cases reported by Mayo Robson. It is important however, to remember that it may not be the hyperacidity alone, but the continuous flow of gastric juice, the hypersecretion so commonly observed in these instances, which supplies the corroding agent.

This hypothesis though plausible is insufficient to explain all the cases thus far observed. If hyperacidity were the potent factor in inducing jejunal ulcers which we suppose it to be, it is more probable that the symptoms of ulceration would appear soon after the operation, especially if we believe that traumatism of the jejunal wall is a contributing cause for such an ulceration. It is the late cases, those which occur two, three, or more years after the operation, that are difficult to explain.

It is interesting in this connection to note that but one case of jejunal ulcer has followed gastro-enterostomy for carcinoma of the stomach. (Lennander.) It may so be that other factors other than hyperacidity tend in exceptional cases to produce the condition.

1. It is observed by some that a contraction of the jejunum below the anastomosis may occur, allowing stagnation and the prolonged action of the gastric juice on the mucous membrane of the jejunum above this point of constriction.

2. Traumatism at the time of the operation may so injure the wall of the gut as to allow the devitalized area to become an easy prey to peptic digestion.

3. The normal circulatory conditions may be so disturbed by the abnormal position and fixation of the loops that form the anastomosis, that areas of local anemia may occur, favoring the formation of ulcers. Thus the loop of the jejunum which passes in front of the transverse colon may be insufficient in length, so that it may become subject to tension, or by kinks in the mesentery itself its blood supply may be impeded.

4. In a few cases jejunal ulcers are probably of infective origin as they occur within a short period after gastrojejunosomy, and are usually multiple. The exact nature of the toxin is unknown.

Paterson represents schematically the etiology of jejunal ulcer as follows:

SCHEME OF CAUSES OF JEJUNAL ULCER

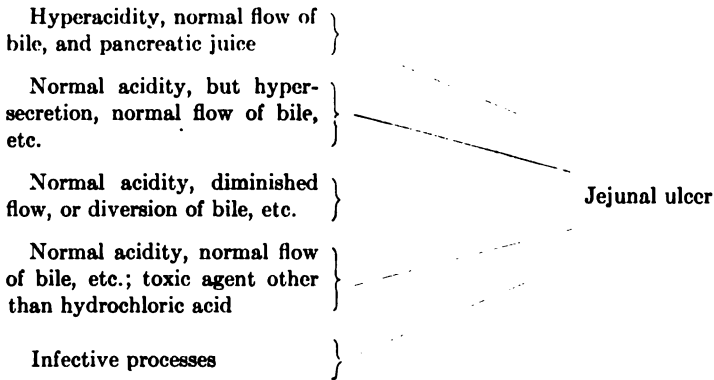
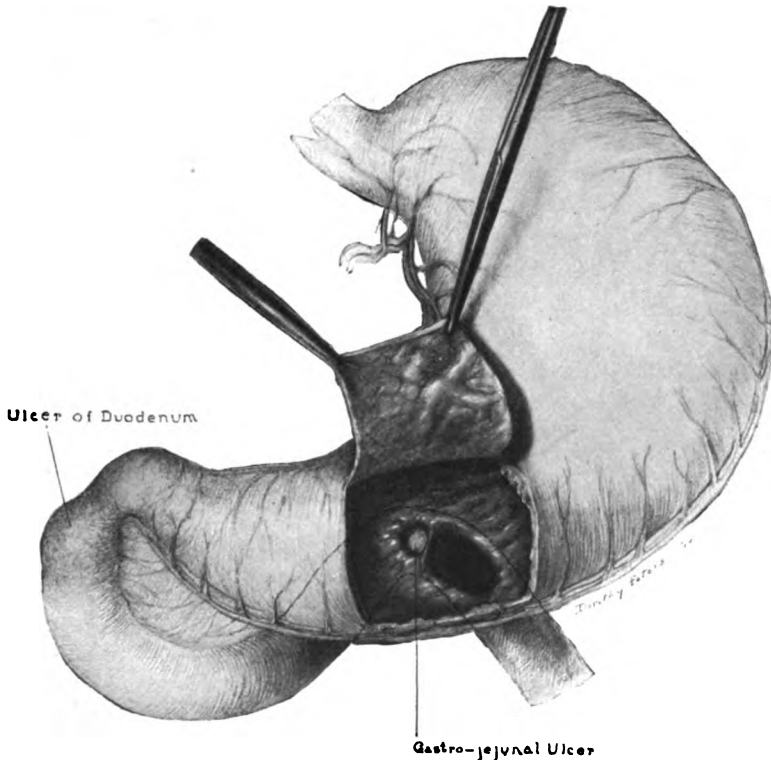


FIG. 34



Gastrojejunal ulcer, the result of infected hematoma. (Mayo.)

Gastrojejunal Ulcers.—Gastrojejunal ulcers, or ulcers occurring at the anastomotic ring, are not infrequently observed. Hitherto they have been included under the general term of jejunal ulcers. A true

jejunal ulcer is rather an unavoidable condition, but the gastrojejunal ulcers are probably due to technical failure in the operation itself. In 1141 gastrojejunostomies done by W. J. Mayo and C. H. Mayo, three such cases are reported: the first being the result of the impaction of a Murphy button, producing a pressure ulcer; the second case occurred as the result of retention of infected suture material; the third was due to infected hematoma in the suture line. The ulcer may be at any portion of the anastomotic ring, or may completely surround it.

Symptoms of Jejunal and Gastric Jejunal Ulcer.—Jejunal ulcers may, like those of the stomach or duodenum, run an entirely symptomless course until they perforate. How many ulcers there are which run a latent course and which heal without ever giving the least evidence of their presence we have no idea, but a review of the cases collected by Paterson leads us to the belief that a diagnosis is made only when perforation or other untoward complications arise. There must be an even larger group which give symptoms so insignificant that the occurrence of the ulcer is unsuspected, or whose clinical course is such that the diagnosis can only be considered possible or even probable.

The period of time between the original gastrojejunostomy and the onset of symptoms varies considerably as is shown in the following table of Paterson's:

Under 1 month	5 cases
2 to 3 months	6 cases
3 to 6 months	7 cases
6 months to 1 year	11 cases
1 to 2 years	8 cases
2 to 3 years	4 cases
Over 3 years	8 cases
Interval not stated	3 cases
	<hr/> 52 cases

It is thus seen that in over one-half the cases the symptoms of ulcer appear within a year and in three-quarters of the cases within two years after the operation. The shortest interval was two days, the longest eight years, while the average in all the cases was twenty months.

The usual history is that after a certain period of freedom from the symptoms for which the gastrojejunostomy was done, the patient will begin to complain again of epigastric pain. The distress is usually greatest two to four hours after meals and is not relieved as a rule by eating, but gradually wears away. Occasionally large doses of soda bring relief. The quality of the food makes no apparent difference in the severity of the pain. The history may closely resemble or may

even be identical with that prior to the operation, and it is probable that many patients who complain of a continuance or a recurrence of their old ulcer symptoms after operation are really suffering from a gastrojejunal or jejunal ulcer subsequent upon the surgical event. The pain, however, usually lacks the clean-cut definition of that of gastric or duodenal ulceration. Gastric analysis, though usually showing hyperacidity, is not noteworthy in any other regard, save that the continual presence of occult blood points toward an ulceration that may involve the anastomotic ring or either loop of the jejunum near the orifice, and this surmise is strengthened if concomitant reaction for occult blood be obtained by examination of the stools. Tenderness, if present, is usually elicited in the median line, or slightly to the left between the navel and the costal arch. The reappearance of epigastric pain or distress some hours after the taking of food in patients in whom gastrojejunostomy has brought relief from their original symptoms for some months, should call most imperatively for close observation, and indicates the necessity for a rigid enforcement of a medical ulcer cure.

With or without an antecedent history of epigastric distress, obvious hemorrhage may occur ordinarily as melenas, occasionally associated with hematemesis. The nearer the ulcer is to the orifice and the more rapid the hemorrhage, the greater is the tendency for the blood to be both vomited and passed by the bowel. Such a case of hemorrhage reported by W. G. Lyle,¹ seen in consultation by the writer, may be briefly cited.

W. B. D., Jr., aged twenty-six years. Patient was well until eight years ago, when he developed gnawing pains in the abdomen, between the ensiform and umbilicus, coming three or four hours after eating and lasting until he ate again. The more he ate the longer the period of relief. The pain continued with periods or remission for seven years, when the ulcer suddenly perforated. He was operated upon, and a perforated ulcer the size of a ten-cent piece was found three-fourths inch from the pylorus, but not causing stenosis. After suturing the perforation a posterior gastro-enterostomy was done, and an entero-enterostomy also performed about four inches below the site of the gastro-enterostomy. Recovery was uneventful, and he was free from all digestive disturbances until six months later, when there was a return of his abdominal pain, with a series of black tarry stools. These symptoms continued for several weeks. Examination of the stomach showed a mild hypersecretion in the fasting condition, of a total acidity of 55, free HCl 30. Test breakfast showed a total acidity of 90, free HCl 60. Stools gave

¹ New York Med. Jour., December 22, 1906, p. 1230.

continuously positive blood reactions. Patient was put under the von Leube treatment, with belladonna and alkalies, and made a good recovery, having had no return of his old symptoms for the past three years.

In certain instances of probable implantment of the ulcerous process at or near the anastomotic opening, either jejunal or gastrojejunal in type, there occur acute exacerbations of more or less constant pain, with nausea and the vomiting of large quantities of acid fluid which contain food remains and usually traces of blood. It is probable that the symptoms arise through inflammatory tumefaction of the tissues forming the edges of the artificial opening into the bowel or from recurring ulcers at the brim of the stoma. Between the attacks the patient may feel perfectly well, eat with impunity, and gain what he has lost. After a certain number of the attacks permanent recovery may result.

The history of a case in which this intermittent course occurred, eventuating in recovery, is as follows:

H. T. C., aged fifty-seven years, came under observation April 27, 1902, with obvious pyloric stenosis and food stagnation. Gastro-enterostomy and entero-enterostomy performed by the late W. T. Bull, two weeks later with uneventful recovery. Remained well for five months eating without discomfort and gaining steadily in flesh and strength. At the expiration of this time he began to suffer pain appearing one and one-half to two hours after his breakfast, lasting until 4 P.M., so that he could eat no lunch. The pain was in the epigastrium, shifting somewhat to the left. After the subsidence of the pain he would feel comfortable enough until 9 P.M., when the pain would reappear and continue throughout the greater part of the night, gradually waning toward morning. On October 30, two weeks after pain began, the patient was examined. Four hours after a breakfast of tea and toast he vomited a large quantity of brown acid fluid. A tube was passed and 800 c.c. of brown acid fluid and ancient food remains were removed. Total acidity 60, free hydrochloric acid 48. The following morning (October 31) the fasting stomach contained 240 c.c. of clear acid fluid without admixture of food remains. The next day (November 1) he had no pain whatever, and the fasting stomach was empty, but on November 2, the pain returned and five hours after a light breakfast 1300 c.c. of fluid and food remains were removed. The patient was put to bed on a milk diet for one week and rapidly improved. Four weeks later (December 8) he began to complain again of heart-burn, pyrosis, and pain just to the left of the median line. At this time he had a number of black tarry stools and became quite anemic. Tube at 7 P.M. withdrew 2 quarts of fluid and old food remains, some of which were eaten thirty-six hours previously. The following morning the fasting stomach

contained 140 c.c. of fluid and food remains containing a large amount of altered blood. Total acidity 58, free hydrochloric acid 29.

December 10, patient was placed on the von Leube ulcer cure, during the first three weeks of which he complained of recurring epigastric pain relieved by food and alkalies. Gastric analysis showed a continuous hypersecretion. After the third week the symptoms entirely disappeared and he remained well for a year, eating everything without distress and gaining in strength. On December 14, 1903, he was chilled while duck shooting, vomited several pints of altered blood, and had a number of tarry stools. His pain returned as before, and on December 28 he was again placed on the von Leube ulcer cure. In this ulcer cure pain and hypersecretion continued until the tenth day, but then subsided and he remained well until April 1, 1904, when his symptoms began again and he had a repetition of his intestinal hemorrhages and was placed again on the von Leube treatment. During this third ulcer cure he complained of severe pains from the tenth to the twentieth day. During this time the stomach contents were aspirated every four to six hours by the tube, the estimated hypersecretion amounting to 35 to 50 ounces a day. From the thirtieth day onward his hypersecretion ceased and with the exception of one slight attack of pain and hypersecretion he has remained well until the present date, a period of nearly nine years.

Diagnosis.—In the majority of the reported cases the diagnosis of jejunal or gastrojejunal ulcer has not been made prior to perforation, the diagnosis being either made at operation or post mortem, so that the literature of the subject is somewhat restricted to the ulcers that actually perforate, those that do not rupture remaining undiagnosed and unreported. Consequently, perforation in the reported cases is of extreme frequency.

Of 52 jejunal and gastrojejunal ulcers reported by Paterson¹ perforation occurred in general peritoneal cavity in 19 cases.

Perforation, limited by adhesions, resulted in inflammatory exudation into the abdominal wall in 28 cases.

Perforation, limited by adhesions, into the colon in 5 cases.

Perforation into General Peritoneal Cavity.—It is interesting to note that of the 19 instances of perforation into the general peritoneal cavity, in only four was there any indication that the result of the gastrojejunostomy had not been entirely satisfactory. Gastrojejunal ulcers are less likely than the jejunal form to perforate into the general peritoneal cavity.

Maylard² has reported an interesting case of two consecutive perfora-

¹ Proc. Roy. Soc. Med., June, 1909.

² Lancet, February 19, 1910, p. 497.

tions of peptic jejunal ulcer following gastrojejunostomy for a perforated gastric ulcer. A similar instance of consecutive perforation is recorded by Battle.¹

The symptoms of perforation do not differ in any material way from those observed in the course of gastric or duodenal ulcerations.

Perforation into the Anterior Abdominal Wall.—There is a group of cases in which the ulcer becomes adherent to the anterior abdominal wall. This happened in 28 out of 52 of Paterson's cases. The ulcer being shut off from the general peritoneal cavity, the adhesions perforate and form a cavity in the substance of the abdominal wall. The history of such an event is that the patient will complain of a severe pain, more or less localized over the upper portion of the right rectus or the left rectus muscle, with tenderness and localized rigidity of that portion of the abdominal wall. A hard tender swelling may be distinctly palpable, often of the size of the fist. The distress is more or less continuous, uninfluenced by the taking of food, but is usually aggravated by any action of the patient which throws a muscular strain on that portion of the abdomen. In certain cases a fistula forms, from which ingested liquids escape.

Perforation into the Colon.—A rarer complication is one in which the ulcer becomes adherent to and perforates into the colon. In these cases after considerable antecedent abdominal pain, usually quite intense, the patient may complain of eructations having the odor of sulphuretted hydrogen, followed by the vomiting of fecal material, or of fluid having a markedly fecal odor. Injections of colored water, such as gentian violet, into the rectum may be drawn off from the stomach upon passing the stomach-tube. In a case reported by Kaufmann² there formed not only a jejunocolic fistula but a gastrocolic fistula as well, with a spontaneous closing of the gastrojejunal anastomoses.

Prognosis.—The prognosis is difficult to determine because the statistics are largely compiled only from the severe cases which come to a secondary operation, but the outlook is always grave whether the operation is done or not. Cases in which the posterior operation has been performed usually give a higher rate of mortality than those who have been subjected to the anterior operation, because in the former instance the ulcer is very deeply seated, and access to it by reason of adhesions is often tedious and difficult.

Treatment.—Improvements in the technique of gastrojejunostomy are responsible for what seems to be a diminishing frequency of jejunal and especially of gastrojejunal ulcerations. The latter form is considered by Mayo to be largely due to a failure of surgical technique.

¹ *Lancet*, 1906, ii, 1246 and 1247.

² *Medical News*, July 8, 1905.

Paterson emphasizes the necessity for a large opening, and the careful application of the inner suture so as to avoid localized necrosis of the tissues.

The necessity for after-treatment of cases of gastrojejunostomy has not received sufficient attention by the surgeons. Patients after this operation are ordinarily allowed to eat anything without restriction, and it seems to be a matter of personal pride on the part of the surgeon that his patient enjoys an unlimited diet within a few weeks of the operation. Such a course of action cannot be too strongly condemned. A patient after gastrojejunostomy should be given rigid rules as to his diet, and if necessary should receive appropriate medical care for at least six months, or until such a time as examination shows that the gastric acidity is normal. The persistence of hyperacidity after gastrojejunostomy is due either to extreme hyperacidity before the operation so that its subsequent natural reduction after the procedure is insufficient to reduce it to normal limits, or because of a too small, ineffective, or defective anastomotic opening. Whether or not indiscretion in diet may induce hyperacidity with a normal anastomotic opening is doubtful. During the early postoperative period, and especially if the patient be known to have suffered from hyperacidity or hypersecretion prior to the operation, repeated doses of bicarbonate of soda or of other alkalies should be systematically given, and the diet should be that of the second or third week of the ordinary ulcer diet.

Stool examination should be made from time to time, and the diet should not be increased should blood reaction be positive.

When the symptoms of ulceration appear and a tentative diagnosis is made, rigid ulcer cure should be at once instigated. Those who have read Tiegel's article and other surgical descriptions of the operative treatment of these cases will be struck by the numerous difficulties which attend such an operation, by their high rate of mortality and often by their complete failure even as a palliative measure. As far as possible, therefore, attempts by medical means should be made to favor the healing of the ulcer, even though, as in the case cited by the writer, the ulcer cure be frequently repeated. It is only when medical means fail that surgical intervention is to be advised.

Perforation is regularly, however, to be treated surgically and without delay. Perforations into the abdominal wall and jejunal colic fistula are, of course, to be considered surgical complications.

FOLLICULAR ULCERATION OF THE STOMACH

Owing to their insignificant size and deep situation in the mucous membrane, the solitary glands of the stomach are less frequently the

seat of disease than are similar glands of the intestine. In certain infective disorders, however, such as typhoid fever, acute tuberculosis and diphtheria, as well as a number of inflammatory conditions of the stomach, these glands become inflamed and undergo necrosis. Such a process results in small ulcers, which are usually about 2 millimeters in diameter, with overhanging edges, scattered over the whole surface of the mucous membrane of the stomach. They seldom extend deeper than the submucous tissue, although it is possible for a small follicular ulceration to extend its area in all directions and present the characteristic form of an acute perforating ulceration. As an evidence of the frequency of follicular ulceration, Fenwick records their presence in 4 out of 10 fatal cases of acute tuberculosis examined by him.

Symptoms.—Occurring during the course of severe infectious diseases, such as tuberculosis or typhoid fever, the symptoms are apt to be obscured by those of the original disease, and the symptoms, if any, that are present are not deemed significant of any gastric disorder. Consequently follicular ulcer of the stomach is an ailment concerning which we possess but little clinical knowledge.

UREMIC ULCERS

Not infrequently erosions or ulcerations are found in certain portions of the alimentary tract in fatal cases of Bright's disease. While the lower portion of the ileum and the upper portion of the colon are the areas most affected, ulcers may be found in the stomach, especially in the pyloric portion and in the duodenum. Such a localization is, however, rare, and in the study of the reported cases, there is often considerable doubt whether the ulcers were secondary to the uremic state, or whether they were independent lesions, merely coexisting with the renal disease. Nevertheless, it seems to be an established fact that such ulcers may occur. In the stomach the lesions are found especially in the pyloric end, while in the duodenum they are usually confined to the first portion. Solitary ulcers are more frequent than are the multiple. They may be at the summit of the *valvulæ conniventes* or in the furrows or the under surface of these folds. They may be only surface erosions, or deep and extensive, so that perforation, hemorrhage, or erosions of the pancreas may occur. The direct causation of the ulcers cannot be satisfactorily explained.

ULCERATION OF THE DUODENUM IN CASES OF BURNS

The fact that duodenal ulceration may occur as a complication of extensive burns of the body has been known for many years. In the

older literature of the subject the name of "Curling's Ulcer" was applied, although it had been described by Long, of Liverpool, two years before Curling's paper was published.

The ulcer may be solitary or multiple, and although the lesion is most conspicuously present in the duodenum, similar ulcerations may occur in the stomach, jejunum, or lower ileum. Of 29 cases reported by Perry and Shaw a single ulcer was recorded in but sixteen instances. The first portion of the duodenum is the favorite seat of selection, more rarely in the first and second, or in the second part. The ulcer may be superficial, or deep and sloughing. Acute inflammation of the duodenum is almost regularly present in the neighborhood of the ulcer.

This complication is supposed to occur in about 6.2 per cent. of extensive superficial burns, and occurs twice as frequently in females as in males, probably because, by reason of the difference in their clothing, women are more frequently and more extensively exposed to burns than are men.

Etiology.—Duodenal ulcer complicating burns is undoubtedly of toxic origin and is comparable with the ulcerations seen in other septic conditions. It is the rule to find the ulcer only when septic processes follow the sloughing of the burnt skin, and the frequency of ulceration in these cases depends upon the frequency in which the suppurative and septic process are present. It has been surmised by some that the ulcer may be due to septic emboli originating in the infected area, producing hemorrhagic erosions in the alimentary tract, which are converted to ulcers by autodigestion in those portions of the duodenum that are most exposed to the corrosive action of the gastric juice.

Symptoms.—In a number of cases the symptoms of duodenal ulcer are either latent or so obscured by those of the burn that the disease is unsuspected and a diagnosis is only made at the postmortem examination. In the majority of instances, however, hemorrhage or perforation occur suddenly as the first indication of the lesion. In 20 of the 29 cases reported by Perry and Shaw, one or both of these symptoms were noted. Hemorrhage is about twice as frequent as perforation and may prove fatal as early as the fourth or fifth day, or as late as the thirty-seventh day after the accident. Its maximum frequency occurs about the end of the second week. Perforation may occur between the fifth and the twenty-first day.

Prognosis.—Recovery is quite exceptional. Death may result from the burns before the ulcer has time to perforate or bleed. In some instances the patient lives a very considerable time suffering from suppurating sloughs and the symptoms of duodenal ulcer before death supervenes.

Treatment.—Treatment is that of the ordinary form of acute duodenal ulceration.

CHAPTER VI

CANCER OF THE STOMACH

CANCER of the stomach is unfortunately a frequent event. Of 1,000,000 hospital admissions, Stockton found that 0.47 per cent. were suffering from this disease. One per cent. of all hospital cases, according to Eichhorst were found affected by the same ailment. These figures are somewhat higher than those observed by the writer. During the years 1904 to 1908 there were admitted to Bellevue Hospital 84,564 medical cases. Of these 143 were diagnosticated as suffering from cancer, a proportion approximately of 1 to 600 patients.

Postmortem statistics show a greater frequency of disease. In 50,000 autopsies compiled by Stockton there were 2000 cancers, a proportion of 4 per cent. Hale White has calculated that 1.5 per cent. of all deaths are attributable to this disease.

The stomach is a very favorite seat for cancer, so that nearly one-half of all cancers are gastric. The number of deaths assigned to this disease has apparently increased from year to year in practically all countries. This fact is less appalling than it seems at first sight when we consider that the increased number of cases reported does not necessarily mean an increase in the actual number of deaths that occur. As more attention is paid to the collection of vital statistics greater accuracy in recording causes for death has resulted, so that the number of cancer cases reported has increased with the increasing efficiency of registration records. Furthermore, refinements in medical diagnosis and an increased number of surgical operations done on the stomach, have increased the number of cases in which a positive diagnosis has been made.

Race.—It was formerly supposed that certain races were less susceptible to cancer than were others. It is somewhat less common in negroes than in the whites, and was supposed to be rare in Egypt and in certain parts of South America. While the manner of living and variations in diet in different races may increase or diminish to some extent the liability to cancer, these differences after all are slight, and in every country in which cancer has been supposed to be rare, the establishment of bureaus for vital statistics has shown that cancer is not as rare as was at one time supposed. In Japan, for example, where it was formerly said that cancer was an infrequent disease, vital statistics

now exist to show that upward of 25,000 deaths from cancer occur every year, proving that the condition is as common as among European races.

Age.—Cancer is a disease having a well-known age incident, the majority of cases occurring between the ages of forty and seventy. In rarer exceptions the disease occurs in early adult life, or even in childhood. Osler and McCrae have reported 10 cases in literature of cancer of the stomach in children under ten years of age, and 13 cases between the ages of ten and twenty. Congenital cases in infants have been described by Williamson, Weiderhofer, and Cullingworth. It is possible that these congenital cases are examples rather of congenital adenoma than of cancer proper. In young children the majority of cases are of doubtful authenticity, as many of them were noted at a time when the finer points of pathology were still obscure. Some of them were apparently instances of congenital adenoma, while others were without doubt examples of the hypertrophic pyloric stenosis of congenital origin.

The influence of age as a factor in cancer is shown in the following table of the author's cases and those from other writers on this subject.

	Stockton combined statistics 7000 cases. Per cent.	Osler. Per cent.	Eichhorst. Per cent.	Welsh. Per cent.	Lockwood 191 cases. Per cent.
10 to 20 years .	0.08	0.0	0.0	0.1	0.0
20 to 30 years .	1.5	4.0	2.0	2.7	2.1
30 to 40 years .	8.8	11.3	8.0	13.3	11.5
40 to 50 years .	18.0	25.3	21.0	24.5	25.7
50 to 60 years .	28.0	32.6	40.0	30.4	36.6
60 to 70 years .	28.0	24.0	26.0	21.0	18.9
70 to 80 years .	14.0	2.6	3.0	6.8	4.7
80 to 90 years .	2.0	0.0	0.0	1.15	0.5

Heredity.—Most of the recent statistical inquiries have tended to disapprove the heredity of cancer, but statistical inquiry is always unsatisfactory because figures can be so arranged, although in perfect good faith, as to lead to widely divergent inferences. The ordinary method employed has been to ascertain what percentage of cancer patients gave a history of direct heredity in their forebears. The general conclusion is that about 10 per cent of afflicted patients show heredity taint. A nearly equal percentage is, however, obtained in the antecedents of those dying from non-cancerous disease.

Bashford, after a careful study of the subjects, has concluded that those dying of cancer have about the same percentage of direct heredity as those dying from all cases whatever, cancerous or otherwise, and Guillot has obtained similar results in his investigations.

An interesting line of inquiry has to do with the frequency of cancer in certain families, which tends to show that while heredity may not be a universal factor, it undoubtedly has some influence in determining malignancy in certain cases. "The family of Madame Z" (reported by Broca and investigated by Lebarde¹) showed 15 deaths from cancer in 26 offspring who attained the cancer age. Of 7 males, only one died of cancer; of 17 females, 14 succumbed to this disease.

Traumatism.—The previous history of traumatism is extremely infrequent in cancer, and its rarity is commented upon by various authors. Occasionally, however, we find a close connection between an injury and the symptoms of malignancy, and it is believed that in these cases the local injury brings out symptoms of a growth previously latent and increases the rapidity of its progress.

Osler reports the case of a man who fell from a wagon while in good health, and for a while was rendered unconscious. The next day he noticed pain in the epigastrium, of a gnawing character, which persisted until his death ten months later. During this time he vomited food every day, but never any blood. Post mortem showed cancer of the pylorus.

In other instances a local injury may originate a traumatic ulcer, which may become chronic and develop malignant degeneration. This sequence is suggested by the following history:

G. B., aged forty-nine years. Patient drinks about 3 pints of beer a day, denies indulgence in stronger liquors. No history of indigestion until two years before his admission to the hospital, when he fell from a step-ladder, landing on his stomach. Shortly afterward he began to complain of pain in the epigastrium coming about half an hour after eating. After four months of this pain he vomited a basin full of black blood. He remained in bed four days, after which time his pain ceased and he remained well for about six months. The pain then returned of a dull aching character; he vomited both food and coffee ground material, and lost forty pounds in weight. He died four months after his admission with typical symptoms of cancer.

Autopsy.—Stomach is adherent to diaphragm, transverse colon, pancreas, and liver. At the pyloric end, one inch from orifice, is an extensive carcinomatous growth extending along the lesser curvature toward the cardia, and along the greater curvature for one-third its length. Growths in liver and intestines and in the seventh rib in the left axillary line.

¹ Quoted by Tysser, Jour. Amer. Med. Assoc., October 29, 1910, p. 1536. Lebarde's article giving the family chart of the family of "Madame Z" will be found in *Revue de Médecin*, 1908, xxviii, 105.

The growth was so extensive that it was impossible at the autopsy to prove its ulcer origin, although the clinical history certainly warranted such an assumption.

Pathology.—Cancer of the stomach consists of an atypical and lawless proliferation of epithelial cells beginning in the glands of the mucosa, invading successive coats of the stomach, spreading to adjacent organs directly, and involving distant parts of the body by metastases through the lymphatic channels or blood stream.

In nearly all cases the carcinoma is primary in the stomach, secondary cancers being comparatively rare. Welch collected 37 cases, of which 17 were secondary to cancer of the breast. Martin states that combined statistics show the proportion to be about 1.1 per cent. *i. e.*, 5 cases out of 440. Fenwick and Fenwick, in their series of 265 consecutive necropsies upon cancer of the stomach, found that 19, or 7 per cent., were secondary to disease of some other organ. This estimate closely agrees with Hale White's 6 to 7 per cent.

Fenwick and Fenwick found that of their 19 cases "no less than 14, or 73.6 per cent., were due to direct extension of the disease from some neighboring organ; that in 4, or 21 per cent., the primary complaint was situated in the upper part of the digestive tract; while in only 1, or 5 per cent., was the gastric affection of the nature of a true metastasis."

The location of the tumor is a question of considerable importance in its bearing upon the symptoms of the condition, and upon the changes in the stomach itself. Until comparatively recently the pylorus was considered by far the most frequent site of the tumor. For example, Welch, in an analysis of 1300 cases, found the pylorus involved in 60.8 per cent., while the lesser curvature showed but 11.4 per cent. and the cardia 8 per cent.

In reaching a solution of the question we are confronted by the same problem as in other gastric conditions; that is, the material obtained at autopsy furnishes unreliable results. The neoplasm is often so far advanced that it is impossible to tell where it began. More recent observations have been made on specimens obtained at operation, and, as a result of these studies, the lesser curvature has come to be regarded as of about equal, if not of greater importance as the site of origin of gastric cancers. Mikulicz and Kausch believe that the lesser curvature is the site of origin in about 40 per cent. of cases, and Boas, in studying 40 cases, found it involved in 62.5 per cent. and the pylorus in 12.5 per cent., practically reversing Welch's earlier figures.

Fenwick, in an analysis of 1850 cases, concludes "that in 79.4 per cent., or in about four-fifths of all cases, carcinoma commences in the comparatively small strip of tissue which extends from one orifice

to the other along the upper margin of the stomach, and that its percentage rapidly diminishes the further we proceed from the pyloric valve." He considers it of relatively little importance whether the cancer begins at the pyloric valve and spreads inward, or develops on the lesser curvature near the orifice and becomes sharply limited by the valve.

The cardia comes next, in order of frequency, the anterior and posterior walls, greater curvature and fundus being less commonly affected.

Occasionally one finds two or more separate growths in the stomach. Thus, in Fenwick's 1850 cases of carcinoma, there were multiple growths in 54, or about 3 per cent. The majority of these cases probably represents some form of auto-infection. Some, however, as for example where there is a tumor at either orifice of the stomach presenting different histological pictures, must be accepted as multiple primary tumors.

Cancer of the stomach presents a varied pathological picture. Considerable confusion arises in the various classifications given, due to the fact that titles descriptive of the form and appearance of the growth are mixed with those indicating the histological picture present. Thus, we see the terms villous, medullary, encephaloid, scirrhus, cauliflower, colloid, adenocarcinoma, spheroidal-cell carcinoma, etc.

It is, therefore, much better, from the standpoint of clearness and of a proper appreciation of the morphology of the tumors, to divide them on a histological basis, adding the various descriptive terms in their proper places.

Following this classification we may recognize three main types of primary gastric carcinoma (Fenwick).

1. **Spheroidal cell or glandular carcinoma.** The cells are spheroidal in shape and similar to those found in the normal gastric tubules. If these tumors are rich in cells, with comparatively little stroma, they are known as medullary, soft, or encephaloid carcinomas; while if there are but few cells in a dense fibrous tissue stroma, the term scirrhus or hard is applied.

2. **Cylindrical cell or adenocarcinoma.** Here the cells are of cylindrical or columnar form, and resemble those found in the pyloric glands.

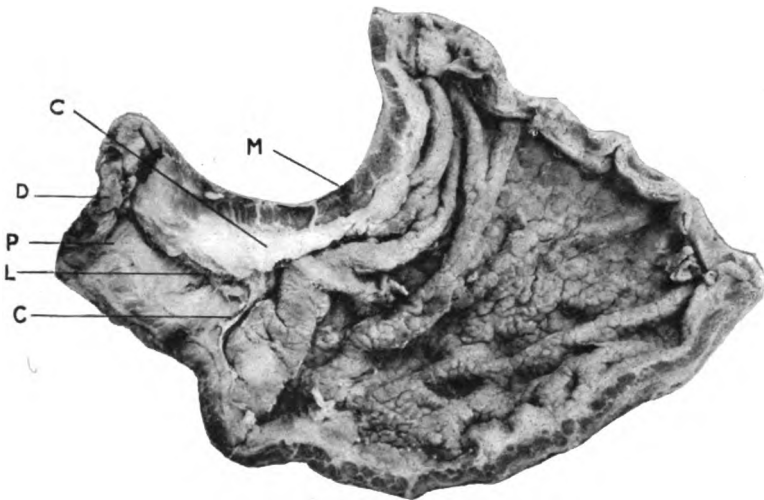
3. **Colloid carcinoma.** In this form, which represents a myxomatous degenerative process of either of the first two varieties, both the cells and stroma may be converted to a greater or less degree into colloid material.

While the foregoing types represent clear-cut differences, it must be remembered that there are various transitional forms which present a more complex picture.

Spheroidal-cell Carcinoma (Glandular).—Scirrhus (Hard) Form.—This type of carcinoma, which is of slow growth and less liable to form

metastases than the other forms, occurs most frequently in the pyloric region. In some cases it may completely surround the pyloric canal; in others it may appear as a diffuse infiltration of the coats of the stomach in this region, with raised edges and depressed centre, resembling a healed scar of a chronic ulcer; while in still other cases one may see at the pylorus a localized, more or less globular tumor, with some cystic or colloid degeneration, and often extensive central ulceration. In all of these forms there is frequently quite marked stenosis of the pylorus, with subsequent dilatation of the stomach. The mucous membrane over the growth is indurated and tough, immovable upon the underlying tissues, may be uneven and show small nodules of tumor tissue. Very often there is superficial or deep ulceration.

FIG. 35



Scirrhus carcinoma of pylorus. Pyloric half of stomach obtained at operation. *D*, duodenum; *P*, pyloric valve; *L*, lumen of the much contracted pyloric canal; *M*, muscularis showing the invasion by the carcinoma, *C, C*. The white, thickened strands of connective tissue are well seen, also the hypertrophy and diffuse carcinomatous infiltration of the muscularis and submucosa gradually decreasing in intensity toward the cardia.

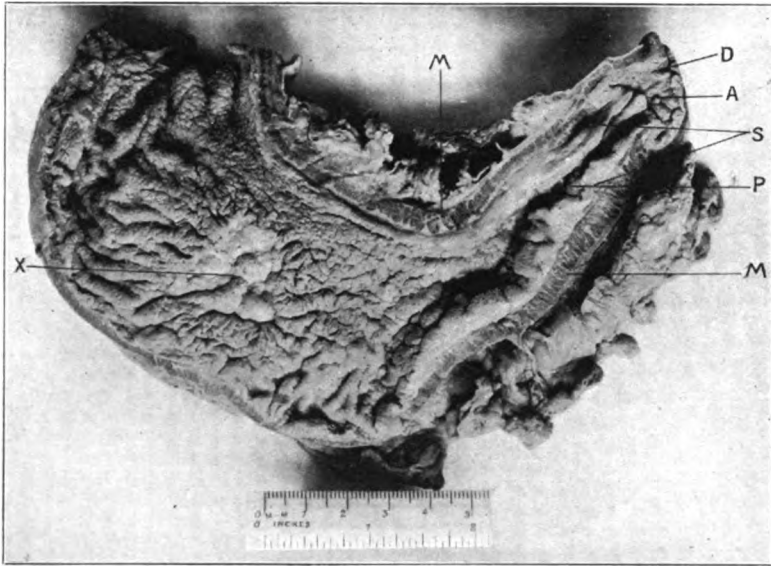
On section of the stomach wall through the tumor, all of the coats are found thickened, especially the submucosa, whose slightly concave, hard surface appears smooth, white, and glistening. The muscularis, especially the circular coat, is considerably hypertrophied, and is traversed by glistening white strands of connective tissue.

More uncommonly scirrhus carcinoma may occur as a diffuse infiltration of the whole stomach wall. This leads to great increase in thickness of the gastric wall, generally most marked at the pylorus, and extreme reduction in the size of the organ. The stomach is

converted into a tough-walled, incollapsible tube, in the extreme cases having a capacity of only one-half to three ounces. The name "leather-bottle" stomach has been applied to this condition.

On section, the submucosa and muscularis are found especially thickened, the induration gradually diminishing from the pylorus toward the cardia. As a rule the indurated mucous membrane shows some ulceration. It may, however, be tough and smooth.

FIG. 36



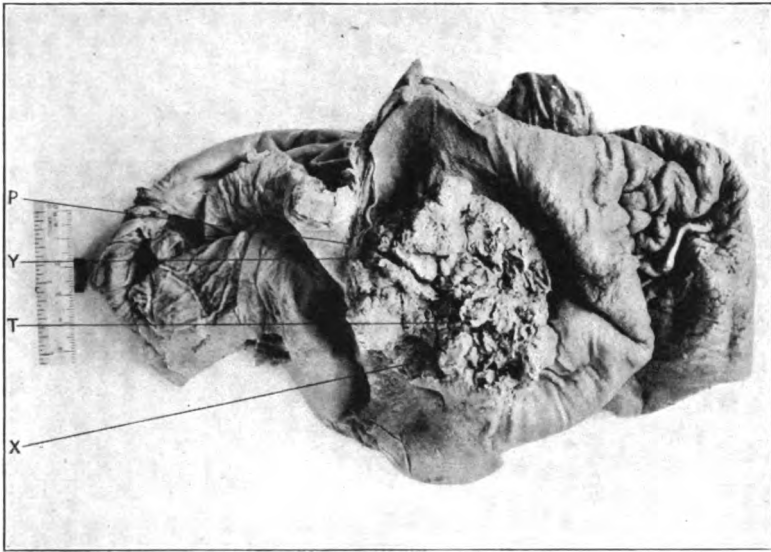
Diffuse scirrhus carcinoma of anterior wall of stomach viewed from behind. The mucous membrane is hypertrophied and the rugæ are stiff and thick. An irregular area of ulceration is seen at *X*. The thickening of the submucosa is well seen at *S, S*. The muscularis *M* is much hypertrophied and is thickly set with interlacing bundles of fibrous tissue. The pyloric canal *P* is narrowed and the capacity of the stomach much reduced. *A*, pyloric valve; *B*, duodenum. (From the Pathological Museum of the Presbyterian Hospital, New York.)

It may be very difficult, macroscopically, to distinguish this diffuse form of scirrhus carcinoma from the condition known as "cirrhosis ventriculi." Even with the aid of a microscope it may require patient search, and even serial sections to settle the nature of the lesion. There are many, indeed, who doubt the existence of a simple cirrhosis ventriculi. Certain it is, that the more painstaking the microscopical examination the fewer are the cases of simple cirrhosis.

Very rarely one may see the growth most marked at the cardiac orifice, or as an annular tumor in the central part of the stomach. In scirrhus carcinoma one frequently sees small tumor nodules of the serous covering of the stomach, and in the peritoneum.

Microscopically, one finds the various coats of the stomach thickened and very rich in connective tissue, which occurs especially in irregularly branching septa of varying thickness. Scattered through the coats, but occurring especially in the thickened submucosa and along the connective-tissue septa in the muscularis are found small nests of spheroidal and atypical epithelial cells. These may occur in small tubules, but more often as solid plugs or strands of cells along the septa, or penetrating between the muscle bundles. They may be more or less degenerated, making it very difficult to tell whether they represent a neoplastic growth or not. The mucosa generally shows the changes of chronic productive gastritis, often with ulceration. It may be invaded by the tumor tissue.

FIG. 37

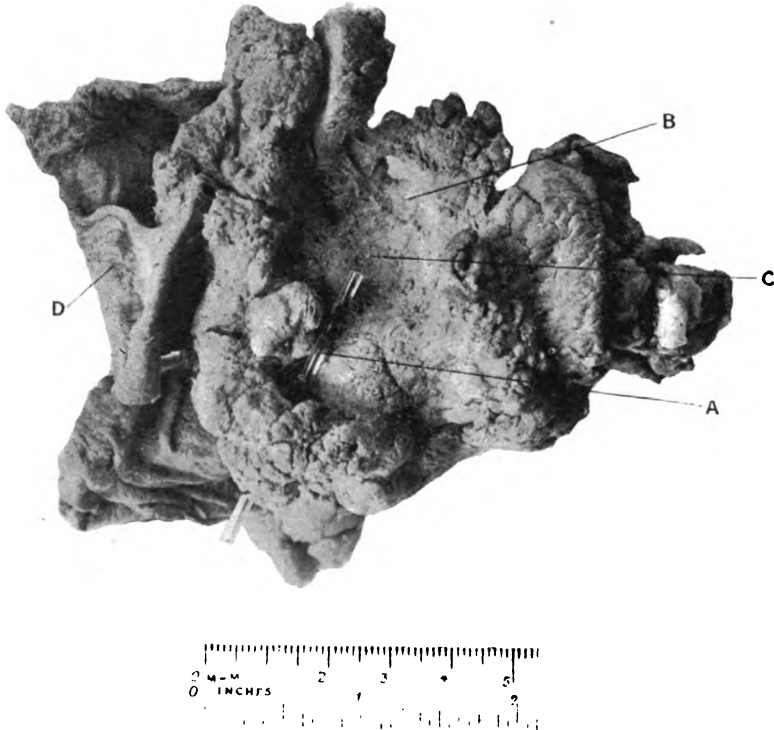


Medullary carcinoma (spheroidal cell) on posterior wall of the stomach at the pylorus. The tumor is of the typical cauliflower appearance. There is some superficial ulceration at X. P, pyloric ring; T, tumor mass, sharply circumscribed. A piece of tissue has been removed at Y for sections.

Medullary (Soft) Form.—These growths appear typically as cauliflower-like excrescences of soft, exuberant, succulent tumor tissue, attached to the stomach wall by a broad base. They are grayish white to pale pink in color, generally show varying degrees of irregular ulceration, have a dirty looking surface or one discolored by hemorrhage, and generally extend deeply into the stomach wall. They grow rapidly, are often the seat of hemorrhage, and form early metastases. On account of the resemblance of the tumor tissue to brain substance this type of growth has been called "encephaloid." As a rule these tumors

appear near the lesser curvature in the pyloric half of the stomach. At times the growth may extend for some distance in the long axis of the stomach, and rarely the cauliflower growth covers practically the whole interior of the stomach, causing great reduction in its capacity.

FIG. 38



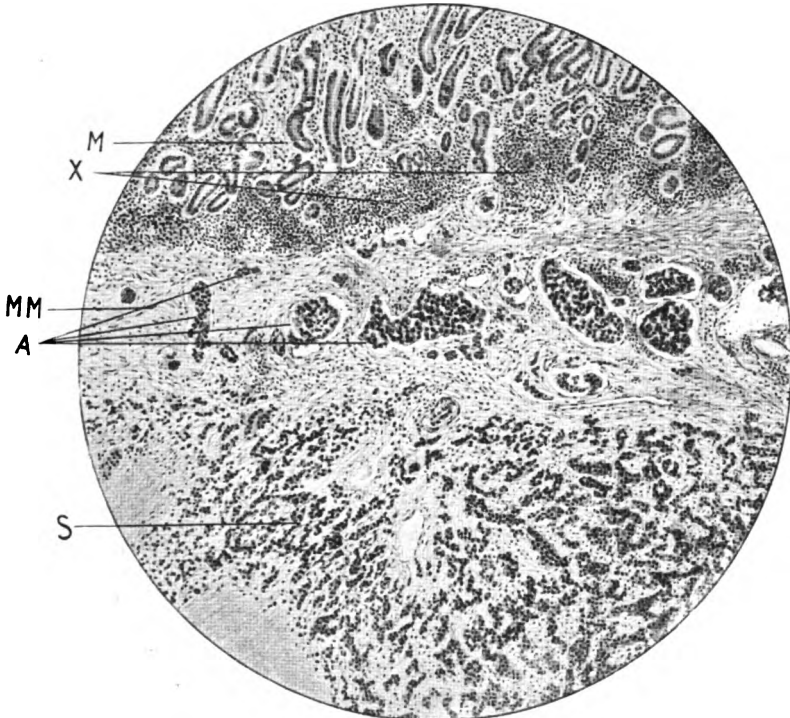
Adenocarcinoma of the papillary-polypoid type which has undergone colloid degeneration. The exaggerated polypoid condition is well shown, one of these exuberant masses forming a bridge of tissue beneath which a glass rod (A) has been placed. Some superficial ulceration is shown at B. The honey-combed appearance of the growth appears at C. D, duodenum. (From the Pathological Museum, Bellevue Hospital, New York.)

Ulceration is very common, at times being so extensive that only a bowl-shaped ulcer, with raised, overhanging, or gradually sloping borders, remains (Aschoff's soft, ulcerating type). Not infrequently one finds other smaller, flat tumor nodules in the vicinity of the large ulcer. These may also form superficial ulcers.

Occasionally, medullary carcinoma infiltrates the walls of the stomach diffusely, causing thickening of the various coats and a hypertrophy of the mucous membrane, which is thrown into firm folds and ridges.

Microscopically the picture is quite the reverse of the scirrhus form. The stroma is scanty, the cell richness great. The alveoli are large, irregular, tortuous, and numerous, and they are filled with oval or spheroidal cells. The mucous membrane is the seat of widely growing tumor tissue of the same type. It shows early ulceration. It is also noticeable that the deepest layers of the stomach wall are infiltrated by the growth at an early date.

FIG. 39



Adenocarcinoma of the stomach. *M*, mucosa showing considerable infiltration, with mononuclear wandering cells at *X*; *MM*, muscularis mucosae, with infiltration of the carcinoma, as at *A*; *S*, submucosa and muscularis, with extensive carcinomatous infiltration.

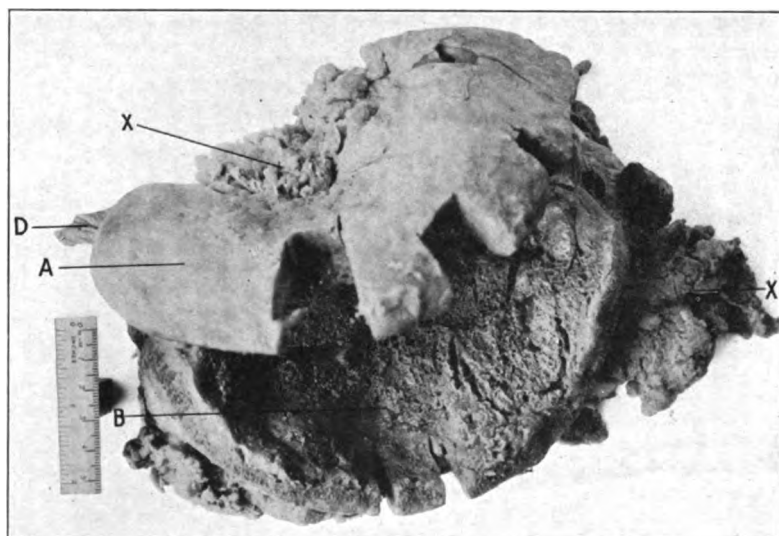
Cylindrical-cell Carcinoma (Adenocarcinoma). — This type is represented by a large, red fungus outgrowth from the mucosa of the stomach, having a broad base and a villous-like surface. While soft in consistence, these tumors are firmer than the medullary cancers, and they are more superficial. They are moderately succulent, very prone to hemorrhage, and often the seat of extensive ulceration. Occasionally they occur in girdle form about the central portion of the stomach, or near the pylorus. Like the other varieties, adenocarcinoma may

infiltrate the stomach walls diffusely. In this form the pylorus is usually thickened and rigidly patent rather than contracted.

On microscopical examination, the tumor is seen to consist of numerous large alveoli of various shapes and sizes in a fine stroma of fibrous tissue, rich in bloodvessels. While in many parts of the tumor the cells may be atypical in shape, one can find alveoli, generally in the deeper layers, lined with typical columnar epithelium.

Colloid Carcinoma.—This type is more uncommon and represents a degeneration form. It may occur late or so early that even the growing edge of the tumor presents a gelatinous appearance. It generally invades all the coats of the stomach, and spreads rapidly to adjacent

FIG. 40



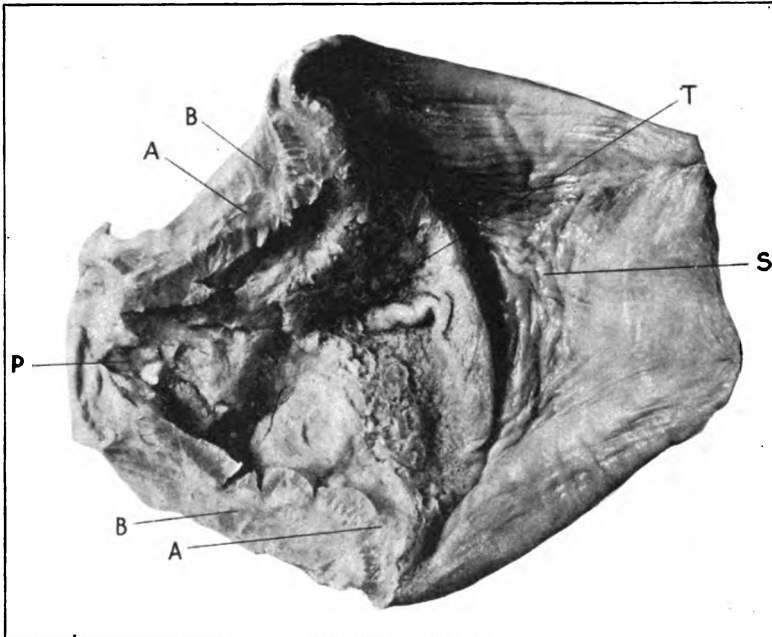
Extensive colloid, showing the increased thickness of the stomach wall. (From the Pathological Laboratory of Mt. Sinai Hospital.)

tissues, especially the omentum and peritoneum. The mucosa is generally thickened and may be very rough, with irregular protuberances in extreme cases resembling polyposis. On section the thickened walls appear honey-combed with larger and smaller alveoli, filled with yellowish or brownish gelatinous material which oozes out on pressure. Colloid carcinoma may appear as a circumscribed mass of light brown color and slimy consistence.

On microscopical examination one sees various sized alveoli, more or less completely filled with the peculiar stringy colloid material. Here and there, many alveoli have almost completely disintegrated, leaving

large spaces filled with colloid and showing irregular loops or strands of epithelial cells. These cells often show granular or fatty changes. The septa may also appear to take part in the degeneration, showing long, slimy processes, or being almost entirely disintegrated. At the margins of the tumor it is generally possible to find some traces of the carcinoma in which the degeneration is taking place. More rarely one finds such degeneration taking place in a scirrhus cancer.

FIG. 41



Colloid carcinoma at the pylorus. The tumor (*T*) is rather sharply demarcated from the rest of the stomach (*S*). The pylorus (*P*) is moderately obstructed. The great thickening of the stomach wall is shown at *A, A*. Note the large increase in tissue outside the muscularis, *B, B*. (From the Pathological Museum, Columbia University, New York.)

In addition to these primary forms one must recognize the rare squamous-cell carcinoma, which is always secondary. These tumors are usually seen at the cardiac orifice, and represent direct extensions of a primary epithelioma of the esophagus. Rarely this type of carcinoma is seen in other portions of the stomach as a secondary nodule caused by transplantation of a piece of tissue from a similar tumor of the tongue or esophagus.

Frequency of the Different Types.—The early figures of Brinton, in which he found that 72 per cent. of gastric cancers were of the scirrhus

variety, 19 per cent. of medullary, and 9.4 per cent. colloid, have been quite generally discarded as valueless. Perry and Shaw, from a microscopical study of 44 specimens of gastric carcinoma, found that 32 were spheroidal cell and 12 were cylindrical cell. Fenwick and Fenwick, in 115 cases, find that 73, or 63.5 per cent., are spheroidal cell, 33, or 28.6 per cent., are cylindrical cell, and 9, or 7.8 per cent., are colloid, and in 41 of the spheroidal-cell cancers, 22 were medullary and 19 were scirrhus.

Changes in the Shape of the Stomach.—These are not so simple as one would expect. Thus in disease affecting the pylorus, it is natural to assume that with the resulting stenosis, there must follow a dilatation of the stomach. But Lebert found that in 20 cases in which the pylorus was obstructed, the stomach was dilated in 13, and contracted in 7; and in 9 cases in which both orifices were free, there was dilatation in 4, and contraction in 5. Fenwick and Fenwick, in 98 cases of pyloric disease, find the stomach "dilated in 52, normal in 10, and contracted in 36."

It is evident, therefore, that there are other factors at work aside from the primary disease. If the neoplasm is situated at the pylorus, and causes obstruction, there is generally an increase in the size of the stomach. When the organ is contracted under these conditions, it is usually due to a diffuse infiltration of the stomach wall by a scirrhus carcinoma. The other forms of diffuse cancer may also cause contraction. In other cases it may be due to the constant vomiting which takes place in these cases late in the disease, and which keeps the stomach constantly empty. Again, adhesions to contiguous organs may cause contraction.

In the cases of rigid patency of the pylorus, the dilatation which often ensues may be explained by the destruction of the muscular tissue in the pylorus, a similar dilatation of the lower esophagus being found when there is a paralysis of its sphincter.

When the tumor causes obstruction at the cardiac orifice, the organ is almost invariably wasted and contracted. Occasionally one sees irregular deformities in the shape of the stomach, such as the various forms of hour-glass constriction, due to a more or less annular growth at some point between the two orifices.

Changes in the Mucous Membrane.—The mucous membrane of the stomach not directly affected by the tumor, usually shows more or less atrophy of the glands, involving particularly the parietal cells, with interglandular round-cell infiltration. This atrophy is probably a secondary change, and not a primary one upon which carcinoma develops, as some observers, notable Hayem and Mathieu, would have us believe.

The various complications which may develop from cancer of the stomach, as well as the metastases which occur, are dealt with in separate paragraphs.

Epithelioma of the stomach is rare, there being but five reported cases in literature.

SYMPTOMS OF CANCER

Precancerous History.—There is no doubt whatever that a certain percentage of chronic gastric ulcers develop malignancy, nor can it be disputed that many such cases of malignancy give a clinical history of a long-standing preceding ulcer. The frequency of such a malignant degeneration and the percentage of cases of malignancy that give a previous ulcer history vary greatly in the experience of different writers. The points to be decided, therefore, are what percentage of gastric cancers arise from ulcer, and second in what percentage of cancer cases is such a history of previous ulcer obtainable.

The earlier writers estimate the frequency of the ulcer origin of cancer as 6 to 8 per cent., basing their conclusions on the results of postmortem examination. Owing to the extensive growth and ulceration of the neoplasm as seen at autopsy, it is often impossible to decide whether signs of previous ulcer are present. It is therefore to the surgeon who has the opportunity to examine during the early stages of malignancy that we must look for accurate data on this subject.

Wilson and MacCarthy,¹ writing from the Mayo clinic, report that of 153 specimens of undoubted carcinoma taken from the stomach at time of operation, 71 per cent. "presented sufficient gross and microscopical evidence of previous ulcer to warrant placing them in a group labelled 'carcinoma developing from previous ulcer.' Eleven other cases (7 per cent.) showed considerable evidence of precedent ulcer, but not sufficient to warrant placing them in the previous group. In 33 cases (22 per cent.) there was relatively small or no pathological evidence of precedent ulcer."

These figures are sufficiently appalling to make us pause. If 71 per cent. of cancers arise from ulcer, excision of an ulcer whenever it is diagnosticated would be a justifiable precautionary measure. It is to be remembered, however, that the Mayo clinic receives, as a rule, only the chronic ulcers which have resisted all forms of medical treatment and which are sufficiently aggressive in their clinical course as to be no longer endurable by the patient. It naturally would follow, therefore, that these figures do not apply to all classes of ulcer which are

¹ Amer. Jour. Med. Sci., December, 1909.

seen by the internist. There is one point, however, that throws some doubts in the writer's mind as to the correctness of these figures. Those who have for years been treating ulcer of the stomach and who for long periods of time have followed up their ulcer cases, do not find that such patients are apt to develop malignancy, at least from a clinical point of view. The writer has traced a large majority of the ulcer cases treated by him in the past twenty years, and has been impressed by the small number of those who have developed malignancy. It is impossible to give accurate figures owing to the difficulty of tracing many of the patients, but his general impression is that in not more than 3 or 4 per cent. of his ulcer cases have the symptoms of malignancy supervened.

The number of patients with cancer who give the history of a previous ulcer is differently given by the physician and by the surgeon. In the Mayo clinic it is estimated that in about 50 per cent. such a previous history is obtainable. Robson¹ writes: "In no less than 59.3 per cent. of cases of cancer of the stomach on which I have performed gastro-enterostomy for the relief of symptoms, the disease having advanced too far for gastrectomy, the long history of painful dyspepsia suggests the possibility of ulcer preceding the onset of malignant disease." This is quite contrary to Robson's² previous belief, for he wrote of cancer six years previously: "It is, however, rare to elicit a history of very old standing stomach disorder; the first evidences of local disease appear suddenly in persons of perfectly sound health and robust digestion."

On the other hand, statistics from purely medical sources indicate that a previous ulcer history is extremely infrequent in cancer cases. In only four of 150 cancers of the stomach reported by Osler was there an ulcer history (2.6 per cent.), and in not one of the four was the ulcer history clean-cut or definite.

Fenwick in his cases found but 3 per cent. admitted a previous ulcer history, while Eichhorst states that in only 2 per cent. of the cancer cases observed in the Zurich clinic could such a history be elicited. The author's experience is as follows:

Of 174 cases of cancer of the stomach in which the history could be completely taken, 148 gave no history whatever of previous indigestion. Thirteen gave a history of indigestion, either most indefinite and not in the least suggesting ulcer, or else of the indigestion due to alcoholism in its typical form, while only 13 gave a history that pointed to previous ulceration. Thus a clinical ulcer history was obtained in

¹ Cancer of the Stomach, 1907.

² Robson and Moynihan, Surgical Treatment of the Diseases of the Stomach, 1901.

only 7 per cent. of the cases. It is, therefore, the experience of the writer that an antecedent history of indigestion in the cancer cases is not more common than in a similar number of non-malignant cases taken from corresponding walks of life, and *that one of the most striking phenomenon of malignant disease of the stomach is the sudden occurrence of dyspepsia in those of cancer age who have previously been free from all indigestion.*

When cancer follows gastric ulcer two clinical types are encountered. In the majority of cases the symptoms of ulcer merge gradually into those of the malignant invasion, the change being accompanied by a falling off in weight, an increasing chloranemia, and by an aggravation and increased constancy of the pain. Emesis rarely affords anything like its former relief. Sudden anorexia is apt to occur. Occult blood is almost invariably present in the stools, and is highly suggestive if it persist in a patient with gastric ulcer who has been for two weeks on a milk diet.

On order that this change to malignancy be not overlooked, ulcer patients should be repeatedly weighed and their stools systematically examined.

In other cases the ulcer symptoms may have subsided months or years previously, without any digestive symptoms in the interval, until the onset of gastric distress that may seem to indicate a recurrence of the ulcer, but which in reality is due to the beginning of a malignant growth.

Local Symptoms.—Pain.—It is improbable that cancer of the stomach can exist without giving rise to local discomfort or pain at some time or another during its course. In many cases, however, the discomfort is so slight, or occurs at such long intervals, that no mention is made of it except under the most searching questioning, and it stands recorded on the history that the patient has at no time suffered from pain or discomfort connected with the digestive act.

Actual pain is absent in a large number of gastric cancers throughout their entire course. In the writer's hospital cases, 174 in number, 21 per cent. were entirely painless. This closely agrees with the Royal Victoria Hospital statistics, in which 22 per cent. of the patients made no mention of pain.

In private practice the proportion of the painless cases is much less, 15 per cent. of the writer's private cases being unaccompanied by any pain whatever, thus closely tallying with Osler's 13 per cent. of a similar painless course.

Pain when present is apt to be a prominent symptom of the disease, although its intensity is not, as a rule, excessive. The pain is usually situated in the epigastrium, and is somewhat less sharply localized

than is the case with ulcer. Radiation of the pain may occur, but is less frequent than in ulcer. In three of the writer's cases the pain was referred exclusively to the left iliac fossa, a localization of pain not uncommon in ulcers involving the lesser curvature near the cardia.

Pain referred to the chest or the lower end of the sternum may occur, usually but not invariably associated with the growths near the cardia. Pain in the left shoulder may occur as an initial symptom, either alone or associated with epigastric distress.

The character of the pain varies more than with ulcer. In a large number of cases (37 per cent. of the hospital series and 21 per cent. of the private cases) no description of the pain is given. This large number of cases implies a certain indefiniteness in the character of the pain. The patient is unable to describe it as accurately as can the patient with ulcer, a fact which is of considerable importance in the differential diagnosis of the two conditions.

When the pain can be described, it is usually of a dull, aching character—an "aching soreness." This pain occurred in 26 per cent. of the hospital cases and in 14 per cent. of the private patients. Pain of a sharp, lancinating character occurs in only a small number of cases (6 per cent. hospital, 5 per cent. private). Pain of a crampy colicky character is not infrequently seen with pyloric situation of the growth, although it has been observed in cases in which the pyloric orifice is entirely patent. Pain described as "gnawing" occurred in only 2 per cent. of the cases.

The relationship of the pain in cancer to eating is rarely as marked as in ulcers, as the pain of the neoplasm shows a far greater tendency to constancy. In the majority of cases there is complaint of a more or less constant pain, some degree of discomfort being almost always present, amounting at times to exacerbations of considerable severity. In a certain number of cases these exacerbations bear no relationship to the taking of food, but in the majority of instances, an increase in the pain after meals is the usual complaint, the duration of time between the ingestion of food and the appearance of the pain varying from a few minutes to several hours.

Cancers of the cardia are usually characterized by pain during or shortly after deglutition. Constant pain, uninfluenced by the ingestion of food, suggests extension to the peritoneum. In these cases the pain is frequently increased by exercise or by deep breathing.

In the series of private cases 30 per cent. complained of pain, or of a "burning distress," appearing two or three hours after meals, relieved by eating or by taking soda. In these cases the differential diagnosis from ulcer would have been almost impossible had it not been for the deductions furnished by the frequent or constant presence of occult

blood in the stools, persisting after the rigid enforcement of milk or liquid diet. A further reference to these cases will later be made.

In the hospital series of cases, this form of pain seemed to be comparatively rare, and the relief to the pain afforded by eating occurred only in a few instances.

It is a diagnostic point of considerable importance that the pain of cancer is less amenable to liquid or bland diet than that of ulcer. In a case of doubtful diagnosis, pain persisting after a week of peptonized milk diet is presumably due to cancer rather than to ulcer. Orthoform, anesthesin, and soda do not relieve the pain in cancer as they do in ulcer. Although remissions or intermissions of the cancer pain are not as frequently observed as in ulcer, it by no means follows that the pain should be continuous. Very frequently patients with cancer, and suffering often to an extreme degree, enter the hospital, and leave with greatly diminished pain or even with no pain at all. Unfortunately many of these cases pass from observation and the diagnosis never becomes established.

Cessation of pain may also occur in cases of pyloric stenosis from the ulceration of an occluding growth at that orifice, thus relieving the stenotic condition. In these instances, however, the cessation of pain is not accompanied by any improvement in the patient's condition, but rather by an increase in his weakness and cachexia.

Cessation of pain may also follow the formation of a gastrocolic fistula. Physical and psychical shocks may in some unknown manner largely influence for the better the pain and distress. It is frequently observed that after operations, even simple explorations without exsection or gastrojejunostomy, the patient's pain and vomiting cease and the general condition improves to an extraordinary degree. This is a point of considerable interest, and should be borne in mind in advising surgical exploration in doubtful cases, in which the actual risk of the operation is slight, but in which the temporary advantages may be extremely gratifying. A remarkable instance of this is the following case:

T. M., aged forty-nine years. Readmitted to hospital September 26, 1905, with the following history: His mother died in late adult life from "dysentery" of six months' duration. Patient is moderately alcoholic, denies syphilis, and says he has never been seriously ill until his present trouble. No history of gastric ulcer can be obtained. Fifteen months ago he began to suffer from severe epigastric pain, more or less constant vomiting, and noticed that in a month he had lost 40 pounds in weight.

One month after the onset of his illness he was first admitted to the hospital. At this time the fasting stomach was empty. Test breakfast

showed 200 c.c. well-digested breadstuff, total acidity 30. No free hydrochloric acid, no lactic acid, Oppler-Boas bacilli, nor blood.

Exploratory operation a few days afterward revealed a cancer mass at the pylorus and pyloric end of the stomach. Gastro-enterostomy was done, but no attempt was made to remove the growth.

Recovery from operation was uneventful, and the patient left the hospital free from pain and vomiting. For thirteen and a half months he worked as a longshoreman, ate everything, gained 50 pounds in weight, and had absolutely no pain or distress whatever, and did not vomit. Two weeks before readmission he began to complain of constant abdominal pain and tenderness, vomited from time to time, and suddenly became exceedingly weak:

Physical Examination.—On his readmission he appeared as a cachectic emaciated man with slight jaundice. There was marked tenderness over the upper part of the abdomen and well-marked rigidity of the abdominal wall. Examination under chloroform anesthesia revealed a large irregular immovable mass in the epigastrium. The growth apparently involved the peritoneum.

His weakness rapidly increased, vomiting and pain were constant and distressing, and he died two weeks after his readmission and four weeks after the reappearance of his symptoms.

Similar instances of temporary relief from pain occur after nervous shocks, as is shown in the following instance.

An elderly lady was seen in consultation suffering from typical advanced symptoms of gastric carcinoma with a large nodular mass in the epigastrium. Her condition was so bad that it did not seem possible that she could live more than one or two days. A few hours afterward the hotel in which she lived took fire, and after many exciting episodes she was finally rescued, clad only in her night-dress, and taken by ambulance to another apartment. From that time she had no pain, vomiting, or distress for over three months, ate everything, and gained daily in flesh and strength. At the end of this period of improvement, however, she suddenly failed, and died two weeks after the return of her symptoms.

Vomiting in Cancer.—Vomiting is one of the commonest symptoms of gastric cancer, occurring in 80 per cent. of the hospital series and in 65 per cent. of the writer's private cases. It is probable that this difference is due to the fact that the private cases are seen earlier in their course, are more carefully treated at the outset, and that dietetic restrictions are more conscientiously carried out.

The most frequent time for vomiting is after meals, the period varying from a few minutes to several or more hours. Cancers at or near the cardia almost regularly cause earlier vomiting after meals than

those of the body of the stomach, while in cancer of the pylorus, late vomiting, with all the characteristics of gastric stagnation, is the rule.

As in the case of gastric ulcer, although the time after meals at which vomiting occurs varies in the different patients, yet each patient has his own period of time at which he is apt to suffer from his vomiting. In gastric cancer, however, this rule is not as sharply defined as in ulcer, the vomiting in many cases being more irregular in type.

The vomiting in cancer is not as frequently followed by relief to the pain as is the case with vomiting in ulcer. The cancer vomiting is usually preceded by uneasiness and nausea, which may be relieved by the emptying of the stomach, but some degree of pain is exceedingly apt to persist. For this reason induced vomiting is rare in cancer as compared with ulcer. In a certain number of cases the pain is worse after vomiting than it was before.

When vomiting once occurs it is more apt to be a prominent symptom than in ulcer, not only more frequent in its occurrence, but more continuous, and lacking the periods of intermission or remission so commonly seen in ulcer. It is, moreover, less amenable to dietetic treatment than in ulcer. Few ulcer cases vomit after a few days of milk diet and alkalies, whereas in cancer, the vomiting frequently persists no matter how carefully the patient is treated. The vomiting in cases of involvement of the pylorus is usually more pronounced a symptom than in cancers situated elsewhere in the stomach. Cessation of the vomiting may occur in these cases, due to the ulceration of the occluding growth and consequent enlargement of the pyloric orifice, or in rarer cases from the establishment of a gastrocolic fistula.

In many cases the disease first manifests itself by acute and severe vomiting. With or without the history of a preceding dietetic indiscretion, the patient is seized by the uncontrollable vomiting of whatever is taken into the stomach, and for days or weeks is unable to retain anything. In a large number of these cases, diarrhea may occur with the onset of vomiting, so that the case closely resembles acute gastroenteritis. The diagnosis of these cases is often one of the greatest difficulty. It is well in this connection to remember that the vomiting of acute gastritis seldom lasts more than two or three days, and is usually amenable to dietetic treatment, whereas the vomiting of this group of cancer cases may continue for days or weeks and is not apt to be materially relieved by careful dieting. A close questioning, moreover, of the cancer cases, will usually bring out the fact that the patient had been losing flesh and complaining of some indefinite symptoms of indigestion prior to the acute onset.

J. P., aged forty-two years, entered the hospital August 9, 1907. Patient has been a steady drinker of beer, and in addition goes

on frequent spree. Was well until two months ago, when he was sick with "some stomach trouble." This occurred after one of his spree and he does not remember very much about the symptoms at that time. Six weeks ago he was suddenly seized with nausea, vomiting, and diarrhea. Since that time he has been able to retain but little on his stomach, as he vomits as soon as food is taken, the vomited matter being green and offensive. The stools have been profuse, frequent, and watery, but he has lost no blood either in the vomited matters or by the bowels. He thinks that in the past three months he has lost about 30 pounds.

Physical examination: Man of medium frame, greatly emaciated, somewhat cachectic. There are a few fine rales, slight dulness, and prolonged expiration at the right apex posteriorly. His patellar reflexes were not obtained. A distinct hard nodular mass but slightly movable is distinctly palpable in the epigastrium. Vomited matters consist of a brownish fluid, together with small quantities of blood without hydrochloric acid, or lactic acid. Patient lived fifteen days after admission vomiting all food or medication given by mouth, and annoyed by a persistent diarrhea. He had no pain or other gastrointestinal symptoms. Death occurred from progressive weakness.

CHARACTER OF VOMITED MATTER.—The character of the vomited matters vary greatly, depending upon the size of the growth, the presence of retrograde changes, such as ulceration or sloughing of the mass, and upon the patency of the gastric orifices. In the majority of cases the vomited matters consist chiefly of food recently taken admixed with mucus, and do not differ materially from those observed in acute gastritis, except that the vomiting extends over a longer period of time. In other instances the correct diagnosis is at once suggested by evidences of food stasis in the vomited matters, and by the presence of Oppler-Boas bacilli and of altered blood.

The vomited matters may be of a putrefactive or gangrenous odor, suggestive of the sloughing of the cancer mass. In rarer instances the vomited matters may be fecal or of a distinctly fetulant odor. This type of vomiting usually occurs with gastrocolic fistula or with the patulous rigidity of the pylorus. It has been also observed with a complicating intestinal obstruction or with peritonitis either suppurative or malignant.

In 16 per cent. of the private cases there occurred occasional attacks of the vomiting of large quantities of brown acid fluid, usually of high hydrochloric acidity, although the acidity may be low. These cases closely resemble those of ulcer with hypersecretion, and from them a diagnosis may at first be impossible.

It is interesting that only one of the hospital series showed this

peculiar form of vomiting, and the only explanation for the apparent rarity of this type of vomiting is in the fact that the patients are discharged, with the diagnosis of gastric ulcer, owing to the widespread misapprehension that gastric cancer is not apt to be found in cases which show high hydrochloric acidity.

In one case observed by the writer copious fluid vomiting containing lactic acid, but no hydrochloric acid, occurred in a patient during apparently robust health.

L. T., aged forty years, previous history unimportant. Had never complained of indigestion until five weeks before his death, when after playing golf all the morning he ate a hearty, hasty lunch, and played the remainder of the afternoon. Following this he complained of a slight sense of fulness in the stomach after eating for several days, and then remained well and strong, eating everything without discomfort.

Gastric analysis at this time showed fasting stomach contains 22 c.c. brownish fluid, total acidity 42, free hydrochloric acid 26. No lactic acid, sarcinæ, or Oppler-Boas bacilli. Blood positive. Test breakfast 125 c.c., well digested. Total acidity 20, free hydrochloric acid 10.

He continued thus until twelve days before his death, when he had a severe gastric hemorrhage, and at the same time felt a mass in his stomach which he says was not there before.

Physical Examination.—A large, well-developed man of normal healthy appearance, denying any loss of weight. Lying transversely across the abdomen on the umbilical level is a mass extending one inch to the right, two inches to the left, hard, insensitive, freely movable and capable of expiratory fixation. The entire umbilical pit is infiltrated and of a stony hardness.

He was at once put on a rigid ulcer treatment. On the fourth day of his starvation he vomited 82 ounces of brownish offensive fluid, although the total liquids taken in the four days amounted to only 20 ounces. Total acidity 56, no free hydrochloric acid, lactic acid strongly positive. Blood reactions well-marked.

This vomiting continued, though not exceeding 16 to 24 ounces a day, in spite of rectal alimentation.

After consultation with the late W. T. Bull, it was decided to perform gastro-enterostomy, which was done on the tenth day of his medical treatment. Although the stomach had been emptied the previous night and the patient had not partaken of any food or liquid during the night, there were drained from the stomach the morning of the operation, 11 pints of the same blood-stained, offensive liquid, rich in lactic though lacking in free hydrochloric acid. The patient died the day after the operation.

Autopsy showed a cancerous tumor the size of a small orange completely surrounding the pylorus, and causing an extreme degree of stenosis of that orifice. The stomach was moderately dilated, its walls were thin and contained a few secondary nodules along the line of both upper and lower curvature. A cancerous nodule was found in the umbilical pit, but no other metastases were present. The wound was clean and aseptic.

In cases in which there is diffuse cancerous infiltration of the stomach, causing a general thickening of its walls with a somewhat diminished lumen, the so-called "water-bottle stomach" or the cancerous form of linitis plastica, vomiting is often characteristic. Emesis occurs soon after eating and in small quantities, as if the stomach could hold only a small amount of food at any one time. It is especially characteristic for the vomiting to depend more upon the quantity than upon the quality of the food, for if more than a certain amount be taken it is at once rejected. Osler has called special attention to these cases, which in his experience are far from uncommon.

Hemorrhages in Cancer.—Hemorrhages from the stomach in gastric cancer may be divided into two groups, visible and occult, these two varieties having exactly the same significance, differing only in degree.

Visible hemorrhage occurs in 25 per cent. of cases, varying in quantity from the vomiting of coffee-ground material to a profuse or even fatal hematemesis. In the majority of cases the bleeding is slight, differing thus from the classical hematemesis of ulcer. The vomiting of brown fluid of acid reaction has been alluded to under the heading of vomiting. The blood may not be vomited, but may pass in the bowel, giving rise to black, tarry stools. In a small number of cases a large hematemesis may be the first sign of the presence of the growth.

M. C., aged forty-four years. Patient has been in the habit of taking three or four drinks of whisky before breakfast and the same number throughout the remainder of the day, besides several glasses of beer daily. Until onset of present illness has never had a day's sickness that he can remember.

Three months before admission to the hospital he suddenly vomited several cupfuls of bright red blood. The following day he was seized with a dull, aching pain in the epigastrium, which was constant and progressively more and more severe and he has grown exceedingly weak. For the past month he has not been able to retain any food or liquid on his stomach except milk taken in very small quantities at a time. No blood in vomitus since outset. Has lost much weight.

Physical Examination. Man of medium frame, much emaciated, looks ill, and very weak. There is slight rigidity in the epigastrium.

In the left upper quadrant is a tender mass, extending 2 or 3 inches beyond the free border of the ribs, freely movable with respiration. Liver and spleen not palpable. No other abnormalities. Test breakfast, total acidity 10, no free hydrochloric acid, no lactic acid, blood or Oppler-Boas bacilli.

Blood Examination: Red blood cells, 3,192,000; hemoglobin, 35 per cent.; leukocytes, 12,000; polymorphonuclears, 95 per cent.; lymphocytes, 5 per cent.

Urine examination shows faint trace of albumin, many hyaline and granular casts.

Death occurred eight days after admission to the hospital from general exhaustion.

It would be difficult at the onset of such a case to exclude esophageal varices resulting from cirrhosis of the liver.

The detection of occult bleeding either in the gastric contents or in the stools has risen to importance only during the past few years, and is of special value in the recognition of latent cases of cancer and ulcer as it serves to distinguish them from neuroses and other benign conditions. The value of the presence of blood as a diagnostic feature depends upon the care with which other sources of bleeding are excluded.

A positive blood reaction in the gastric contents not infrequently occurs in normal cases from the slight traumatism caused by the passage of a stomach-tube, no matter how soft and pliable it may be, nor with what apparent ease it is passed. Blood in the vomited matter may arise from the stomach or pharynx as the result of the muscular effort of vomiting. A positive test is not of value if rare meat or beef juice has recently been ingested.

For the detection of blood in the stools even greater precautions should be taken to avoid error, as the sources for the bleeding are numerous. The technique and the sources of error in the test for occult blood both in the vomited matters and in the stools are given under Ulcer.

Of the 75 per cent. of the writer's patients with cancer who gave no definite history of hemorrhage, 60 per cent. gave a positive blood reaction in the gastric contents. In many of these cases only one examination was made, and it is probable that repeated examinations would show that almost every patient with gastric cancer will give from time to time evidences of occult bleeding.

The frequency of occult blood in the stools is equally as high as that in the gastric contents.

Positive reactions were found by the writer in nearly every case of cancer observed in which *repeated* examinations for occult blood were made. The remarkable frequency of positive reactions has been found by other writers. In 12 cases enumerated by Hale White 10 gave

steadily positive results, 2 gave reactions from time to time. Joachim found positive reactions in 17 out of 18 cases, while Hartman had positive results in 14 out of 17 cases. Boas found positive reactions in 107 out of 128 cancer patients.

The practical rule should, therefore, be to make frequent, even daily examinations of the stools of those whose clinical history is suspicious of cancer, as well as in those patients of adult years who without adequate cause become anemic and lose flesh and strength.

It must further be emphasized that in every *ulcer cure*, even when the diagnosis of simple ulcer seems evident, not one, but a series of such examinations should be made, and further, that in such a case recurring positive blood reaction in the stools during the milk diet period of the ulcer cure, especially if accompanied by a continuance of gastric discomfort should seriously suggest the advisability of an exploratory laparotomy. These points are well brought out in the following history:

W. L., aged thirty-five years, was well until one year ago. He then began to suffer from gnawing pain in the epigastrium two hours after eating, relieved by eating or else gradually wearing away. Four weeks ago he vomited a small quantity of blood. No other vomiting attacks have occurred during his illness. Aside from a tender spot below the ensiform, the physical examination is normal. His gastric analysis shows the fasting stomach empty, test breakfast normal except for a slight increase in its total acidity. He was put on the von Leube ulcer cure and it would have been considered that the results were favorable except that he remained slightly anemic and the stools on a milk diet showed positive reaction for occult blood. No improvement being noted within the first month of his treatment, an exploratory laparotomy was done, and a carcinoma ulcerated on its surface was found in the lesser curvature, not involving the pylorus. This growth was inoperable. Death occurred five months afterward from hemorrhage.

General Symptoms.—One of the most significant signs of the beginning of cancer of the stomach is a *loss of appetite*, especially for meats and other albuminous food, which may appear suddenly, and which often is so extreme that the patient has a positive abhorrence of all kinds of food. This symptom appearing in a patient of cancerous age who has previously paid little attention to his stomach should never be lightly cast aside, but should call for an examination of the stomach, both in the fasting and in the digestive state. The appetite may improve with careful dietetic treatment of the patient or by judicious lavage, and is apt to return after gastro-enterostomy or even after a simple exploration. There are cases of cancer that run their course throughout with a normal or excessive desire for food. In many

instances, especially when a gastric analysis show a hydrochloric hypersecretion, the patient may be constrained by reason of the discomfort to eat every few hours, as if he were suffering from a duodenal ulcer.

Blood.—Anemia is a symptom common to the majority of cases of carcinoma, although when there has been considerable loss of fluid by vomiting or a decrease in the intake of liquids, the blood becomes somewhat concentrated and may show a normal or an increased number of red blood cells, with an increasing high hemoglobin percentage. In the majority of cases chloranemia is present. The number of red cells noted in the writer's cases is shown in the following table of percentage:

Under 1,000,000	1.8 per cent.
1,000,000 to 1,500,000	7.0 per cent.
1,500,000 to 2,000,000	3.6 per cent.
2,000,000 to 2,500,000	10.0 per cent.
2,500,000 to 3,000,000	10.0 per cent.
3,000,000 to 3,500,000	26.0 per cent.
3,500,000 to 4,000,000	3.6 per cent.
4,000,000 to 4,500,000	19.0 per cent.
4,500,000 to 5,000,000	10.0 per cent.
Over 5,000,000	8.7 per cent.

The hemoglobin is relatively lower than is the reduction in the number of the red cells, so that the color index ranges between 0.5 and 0.7. This is an important point in the differential diagnosis between cancer and pernicious anemia, as in the latter disease we have a higher color index and a greater reduction in the number of the red cells. The usual count in carcinoma is between 2,500,000 and 3,500,000, rarely under 2,000,000, whereas in pernicious anemia counts of less than 1,500,000 are not unusual. It has been said that in carcinoma red cells do not fall below 1,500,000. Five of the writer's cases disproved this statement. The following are the figures:

Cases.	Number of red cells.	Hemoglobin.
1	400,000	20 per cent.
2	1,200,000	18 per cent.
3	1,280,000	16 per cent.
4	1,292,000	20 per cent.
5	1,345,000	18 per cent.

In cancer the count is not as low as would be supposed judging from the appearance of the patient. We may have a fair blood count with an anemia and cachectic appearance. The diminution of the red cells does not keep pace with the cachexia, whereas in pernicious anemia the

cachexia does not keep pace with the diminution of the red cells. In doubtful cases ophthalmic examination may be of service, as punctuate retinal hemorrhages are very infrequently found in cancer, while they are quite common in pernicious anemia.

Failure in Nutrition.—Failure in nutrition may be an early and suggestive symptom, and is especially marked when the cancer involves either orifice of the stomach. In the extraostial form when neither orifice is obstructed, changes in nutrition are less marked. In many instances even of pyloric cancer remarkable gain in weight has followed careful attention to the patient's diet or the judicious washing of the stomach. Improvement in weight and in general nutrition usually occurs after gastro-enterostomy, or may even follow a simple exploration of any nervous or physical shock. The gain in weight in such postoperative cases may be very considerable, occasionally amounting to 50 to 60 pounds. A sudden increase in weight, without any change in the patient's treatment, or any other obvious reason, may indicate effusion of fluid into the pleural or peritoneal cavity or a rapid invasion of the liver. Unexplained loss of weight with failure in the appetite of those of cancer age which cannot be satisfactorily explained, should excite our suspicions of the beginning of malignancy.

Fever.—Fever is present in about one-half the cases at some time or another during their clinical course. The temperature curve may show marked irregularity, the fever may be low and continuous or there may be sudden high elevations to 104 or 105 of short duration for which no cause can be ascribed. In other cases the temperature may assume an intermittent curve, the elevations being preceded by chills and followed by sweating, resembling the temperature chart of malaria or of septic absorption. The rise of temperature in cancer may be due to septic absorption, appearing as a late event during the cachectic period, or it may indicate the presence of complications, of which bronchopneumonia is perhaps the most common.

Many of these high temperatures in late cancer may be explained by the finding at autopsy that the stomach has perforated and formed a false communicating cavity limited by some of the adjacent structures.

Pruritus.—Pruritus is often severe, and itching of the skin in elderly people who show no albumin or sugar in the urine should excite our suspicions.

Urine.—The urine is frequently diminished in amount, owing to the frequent vomiting, and to the diminished intake of foods and liquids, and is most marked in cases of pyloric obstruction. In about one-quarter of the cases the urine contains albumin in small quantities and may occasionally give the reaction for albumose. The ethereal sulphates and indican are usually increased. In patients with pyloric

obstruction who secrete from the stomach large quantities of liquid which is either vomited or drained through the tube and in those cases in which an insufficient quantity of food results from their disease, acetone and diacetic acid, as in all starvation cases, may be present in the urine.

Coma.—The sudden onset of coma with heart weakness, the so-called "coma carcinomatosum," is a rare terminal event in the course of cancer of the stomach. Its exact cause is not accurately known, although it is supposed to be a form of acidosis. It may be accompanied by tetany.

Clinical Types.—It is convenient in describing the clinical types of cancer of the stomach to divide them into *four groups*:

1. Those cases with predominant general symptoms.
2. Those cases with predominant local symptoms.
3. Those cases in which both general and local symptoms are present, comprising the great majority of the cases.
4. Those cases in which the symptoms due to metastases predominate.

I. Cases in Which General Symptoms Predominate, Local Symptoms Slight.—*Dry Shrivelled Type.*—While this type may be encountered in all ages, it is far more commonly in aged subjects, and is especially frequent in almshouse and asylum practice. The patient simply becomes more and more run down and debilitated, and dies exhausted without at any time presenting obvious symptoms of gastric disease, although careful questioning usually elicits the history of painful dyspepsia at some time or another during the course of the disease. It is not uncommon for the local symptoms to be more marked at the onset than later in the disease when the patient is wearied by the ravages of disease. Physical examination may or may not reveal the presence of a neoplasm, but a contracted scaphoid abdomen can be noted in a sufficient number of cases to afford a suggestive clue to the diagnosis. Anemia is seldom marked in this group of cases, many being shrivelled up subjects who give a relatively high blood count. In one dried-up old lady in the writer's care, the blood count shortly before death was red cells 6,080,000, hemoglobin 98 per cent.

Anemic Type.—In other cases the clinical type presented is one of anemia, at first resembling ordinary secondary chloranemia, later approaching the pernicious form in many of its essential features. The onset of an unexplained secondary anemia in those of the cancer age, especially when associated with anorexia and loss of weight, should excite apprehension even in the absence of gastric symptoms or definite physical signs.

Intestinal parasites must be considered a possibility in these obscure anemic cases.

II. Cases with Predominant Local Symptoms, General Symptoms not Marked.—In this group the local symptoms appear early and simulate those due to a variety of acute gastric disorders. Boas estimates that 25 per cent. of the cancer cases begin thus acutely. An abrupt onset is especially frequent in the cancer of young subjects. The more careful we are, however, in obtaining an accurate clinical history, not only from the patient, but from the family and friends as well, the more frequently do we find that in cases in which the onset apparently begins with acute local symptoms, that there has been a gradual loss of flesh and an increasing deterioration of health prior to the appearance of these abrupt symptoms.

In a number of cases, however, which the writer places at about 10 per cent., local symptoms are abrupt and severe without previous appreciable change in the patient's general condition.

Vomiting may appear early and for a considerable time may continue to be the only symptom of importance. In these cases, to which reference has already been made, the diagnosis is often erroneously made of gastritis. When one reflects, however, on the relative infrequency of vomiting in gastritis there is little excuse for such an error, in the great majority of instances.

When the vomiting is characteristic of *hypersecretion*, however, the difficulties of diagnosis are much increased. Such an onset occurs especially with pyloric cancer and is not uncommon. The clinical history closely resembles that of ulcer. The patient usually complains of pain one or two hours after eating, often relieved for a time by eating or by taking soda. From time to time there occurs vomiting of large quantities of acid fluid, often of intense HCl acidity, as in pyloric ulcer. During the intervals of the hypersecretion—vomiting, gastric analyses may show evidences of food stagnation, though usually of slight degree. As these symptoms occur usually early in the course of the disease the nutrition and strength are well maintained, and the case is regarded as ulcer without any suspicion as to its true nature.

The behavior of the case during the ulcer cure usually indicates, however, that there is something more than a simple ulcer that we are treating.

It should arouse our suspicion of malignancy in any case of ulcer undergoing treatment,

1. If occult blood be present in the stools during the milk diet period of the ulcer treatment.
2. If pain, nausea, or vomiting persist after the first ten days of the ulcer cure.
3. If the patient fail in strength or become progressively anemic after the second week, or to lose weight in spite of a sufficient diet.

If any of these indications of malignancy arise an exploration should be thoroughly considered. The following case illustrates the foregoing points:

G. M., aged forty-two years. Well until nine months ago, when he began to complain of pain in the epigastrium two or three hours after eating; relieved by eating. He felt well otherwise. Two months ago he developed nocturnal vomiting, the vomited matter consisting of food remains of his dinner, with large quantities of fluid of an acid taste. After vomiting he would feel absolutely comfortable.

Examination showed slight tenderness at the costal angle.

Fasting stomach, 35 c.c. fluid, few starchy food remains found microscopically. Total acidity 46, free hydrochloric acid 28.

Test breakfast, 45 c.c. well digested—no hypersecretion. Total acidity 50, free hydrochloric acid 20.

The diagnosis was made of ulcer at the pylorus, and the von Leube treatment was started January 5. On the third day of total abstinence from food and drink he vomited 21 ounces of dark green fluid, total acidity 74, free hydrochloric acid 56. On the seventh day (taking only small quantities of milk and vichy) vomited 8 ounces of the same acid fluid. On the ninth day, at 8 P.M., vomited 16 ounces fluid, total acidity 114, free hydrochloric acid 66; at 10 P.M., vomited 15 ounces fluid, total acidity 98, free hydrochloric acid 58. At 5 A.M. the following day vomited 16 ounces fluid, total acidity 72, free hydrochloric acid 54. From this time on, under bismuth subcarbonate, bicarbonate of soda and atropine he had no further vomiting attacks, and the distress in his stomach practically ceased. His stools, however, gave a constant positive reaction for blood, and he continued to lose weight in spite of an adequate amount of food and the most tonic regimen.

Because of the diminishing weight and the constant presence of blood in the stools, and a slight return of gastric distress an exploration was performed May 4. A carcinoma the size of a lemon was found at the pylorus without evidence of previous ulceration. There were no adhesions and apparently no glandular involvement.

In other cases *pain* is an early feature and may exist for a considerable time without other manifestations of disease. In these cases careful examination should be made for chronic appendicitis, for concealed hernia, for epigastric hernia of the linea alba, and for disease of the gall-bladder. The diagnosis from the visceral pain of arterial sclerosis is frequently difficult.

In rarer cases repeated *hemorrhages* may occur as the only manifestation of disease, rendering a diagnosis from cirrhosis of the liver or from ulcer almost impossible.

III. Cases Giving Both Local and General Symptoms.—Although the general symptoms are the same in kind irrespective of the exact location of the growth in the stomach, local symptoms vary greatly according to the location of the part involved. We distinguish clinically between

1. Growths at the cardia.
2. Growths at the pylorus.
3. Growths not involving either orifices.

Growths Involving the Cardiac Orifices.—Painful digestion is a characteristic symptom. The pain is apt to occur at once or very shortly after eating, and is often very intense and distressing. Regurgitation of food is not uncommon, but actual vomiting does not usually occur.

The stomach-tube is usually arrested at about 40 to 42 cm. from the dental arcade, its tip on withdrawal may be coated with pus or blood, and in the eye of the instrument may occasionally be found fragments detached from the neoplasm. These signs and symptoms are indistinguishable from those of esophageal cancer.

Stagnation of food in the stomach does not ordinarily occur with cardiac growths, although should the process extend below the lesser curvature, a motor insufficiency from "infiltration rigidity" may result. In such cases, however, food retention rarely becomes a noticeable feature unless the growths be sufficiently widespread to involve the pylorus by its extension. The position of the tumor under the costal arch renders it inaccessible to palpation unless the growth extends to the lesser curvature with a downward or vertical displacement of the stomach.

Growths at the Pylorus.—Pyloric implantation is the usual clinical type of cancer of the stomach, and requires but a brief mention here as the symptoms have been previously described in detail.

Pyloric obstruction is the prominent feature, vomiting is common, not only of what has recently been eaten, but of the food that has remained in the stomach for an abnormal period of time, or of the copious acid fluid of the hypersecretion type. The physical signs of a tumor are usually present. The diagnosis of this pyloric type of cancer is fraught with fewer difficulties than when the cancer is elsewhere situated.

Growths not Involving Either Orifice.—Extraostial growths are usually situated in the lesser curvature. Such a seat of selection was formerly regarded as infrequent, but of late it is thought that 25 per cent. to 40 per cent. (Mikulicz) of all gastric cancers arise in this situation.

Unless there be extensive infiltration of the stomach wall food-stasis does not occur. Rigidity from infiltration may, however, allow of a moderate motor insufficiency. Should involvement of the pylorus

occur, food-stasis becomes evident. In cases of infiltration of the wall of the pyloric antrum, although no actual distinction to the outward passage of blood may exist, we have a rigid patency, as shown by the paradoxical combination of pyloric narrowing and of pyloric insufficiency. There may be at the same time food retention and duodenal regurgitation. In some cases of cancer of the lesser curvature cardiospasm may be an initial symptom, although the cardiac orifice is free from infiltration by the growth.

The diagnosis of extraostial cancer is often made with difficulty, as the nutrition is frequently well preserved and the local symptoms may lack definite and distinctive characteristics.

Those Cases in Which the Symptoms Due to Metastases Predominate.—It not infrequently happens that a cancerous peritonitis with ascites occurs during the course of gastric cancer and draws our attention away from the primary disease. The distention of the abdomen and the difficulty in making a thorough examination by reason of pain and rigidity obscure what physical signs there might be of gastric growth. Gastric symptoms are present in more or less severity, but these may be misinterpreted as due to the peritonitis. It is in these cases that the diagnosis of tuberculous peritonitis is most frequently made. Less frequently metastases in the lungs and pleura give a group of predominant pulmonary symptoms. A careful study of the case should, however, enable us to arrive at a correct diagnosis. In other cases gastric symptoms are not marked until the advent of such a complication as extends to and perforates into the colon, or a perforation of the stomach wall with its attendant peritonitis.

Extensive deposits in the liver may give the history of cancer of the liver with gastric symptoms well in the background. It often happens that when a primary cancer of the stomach is complicated by metastases in the liver, the growth of the former becomes arrested and the symptoms become relatively quiescent.

METASTASES AND INVASION OF OTHER VISCERA

Carcinoma of the stomach almost always extends beyond the confines of that organ before death to invade contiguous or more remotely situated parts of the body. Such a secondary involvement may occur in two ways: (1) By direct invasion; (2) through the lymphatic or blood stream.

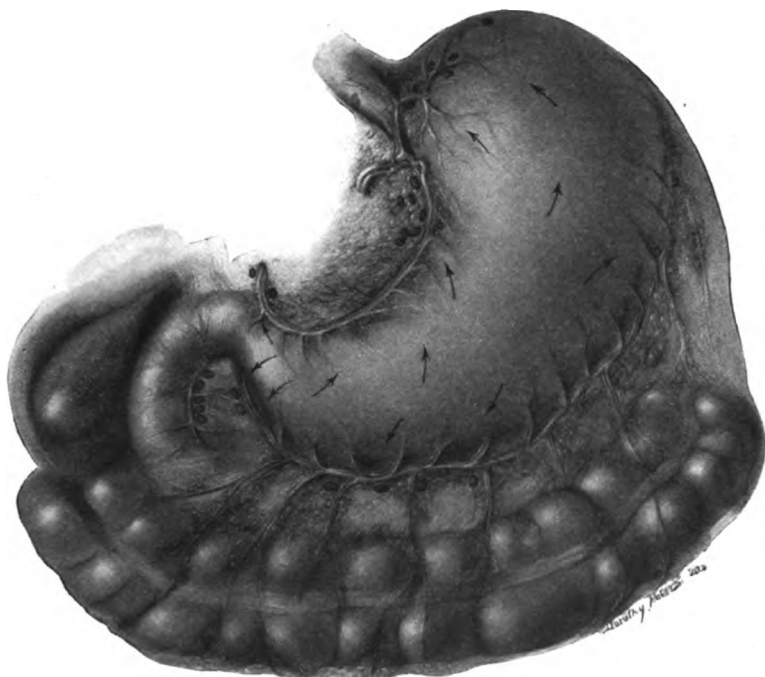
By Direct Invasion.—The growth may extend by direct lines of invasion through bridges of fibrinous adhesions, diaphragm, pancreas, colon, peritoneum, or the abdominal wall.

In 131 of Fenwick's cases:

Pancreas directly invaded in	16.7 per cent.
Liver directly invaded in	13.7 per cent.
Colon directly invaded in	5.3 per cent.
Spleen directly invaded in	3.7 per cent.
Esophagus directly invaded in	4.5 per cent.
Duodenum directly invaded in	1.5 per cent.

As rarities, direct implication of the kidney and the suprarenal glands have been recorded.

FIG. 42



Anatomy of the stomach with special reference to the bloodvessels and lymphatics. The arrows indicate the direction of the lymphatic flow. (W. J. Mayo. Drawings by Dorothy Peters.)

Carcinoma of the pyloric region rarely extends into the duodenum, owing to the lack of continuity of the submucous and muscular coats at the line of fusion, whereas, growths at the cardia are prone to spread to the walls of the esophagus.

Infiltration occurs frequently in the scars from former operations done for attempted relief of the disease. Of 8 such scars in 220 cases of cancer of the stomach in the writer's series 6 were infiltrated.

Infection through the Lymphatic and Venous Channels.—This is the most common mode of extension and is the method of transmission by which metastases form in parts of the body that are not directly in contact with the primary growth by a process of embolism.

Involvement of the *perigastric and retroperitoneal glands* occurs in 85 per cent. of the cases according to Cuneo, who also states that an apparently normal appearance of a gland does not prove that it is innocent of malignancy. This explains why Cuneo's figures are higher than those of purely clinical observers, and for the same reason they are apparently more accurate.

The importance of this glandular metastases is well understood by the surgeon of today in his endeavor to remove these outposts of malignancy as radically as is feasible at the time of operation.

The *supraclavicular glands*, especially those located at the junction of the thoracic duct and vena cava are involved in many cases and may be distinctly enlarged. Such a glandular enlargement is palpable in about 4 per cent of cases, although some observers make this occurrence one of greater frequency. Eichhorst, for example, claims that enlargement of the cervical glands occurs in 21 per cent. of cases of cancer of the stomach. The left-sided glands are more usually implicated, and it is a common routine practice in suspected cases to examine the base of the neck, on the left side only, forgetting that the glands of both sides are frequently involved, and occasionally the glands on the right side may alone be involved.

In 17 cases in which the supraclavicular glands were involved Hosch¹ reports that those of the left side were involved alone in 13, those of both sides in 2, and those of the right side alone in 2. In doubtful cases if tuberculous and syphilitic adenitis can be excluded, this authority advocates excision of the enlarged gland for diagnostic purposes.

In 435 cases of upper abdominal carcinoma reported by Palmer of the Mayo clinic, there were 18 cases (4 per cent.) of supraclavicular gland enlargement. One case was right-sided, one right- and left-sided, the remainder were left-sided alone.

Other glands such as the bronchial and mediastinal may be similarly affected.

According to Stockton, of 2156 cases of carcinoma of the stomach:

The liver was involved in	33.3 per cent.
The peritoneum and intestines were involved in	27.6 per cent.
The pleura and lung were involved in	7.3 per cent.
The pancreas was involved in	7.6 per cent.

¹ Mitteil. a. d. Grenzgeb. d. Med. u. Chir., xviii, Heft 3, 489.

According to Fenwick, the frequency of the rarer metastases is: kidneys 4 per cent., heart 2.3 per cent., ovaries 2.3 per cent., spleen 2 per cent., bones 2 per cent., uterus 1.5 per cent., brain 0.7 per cent. Involvement of the brain is almost invariably associated with a tumor of the lung.

The fact that the veins of the stomach enter directly into the portal system is sufficient to account for the inordinate frequency of metastatic deposits in the liver. These vary greatly in size and number, and their development is often excessive when compared with the size of the original tumor. Fenwick states that metastases in the liver occur with relative infrequency with cancer of the pylorus causing stenosis at that orifice.

Ascites is not uncommon with cancer of the stomach, and was present in 6 per cent. of the writer's cases. The effusion may be due to portal congestion by pressure of enlarged periportal glands, but the usual cause is to be found in cancerous peritonitis. In one of the writer's cases ascites was due to subacute peritonitis from the perforation of the duodenum which had been invaded by the extension distally of a primary pyloric growth. In the ascitic fluid cells showing well-marked mitoses may be found and are of considerable importance in diagnosis. The possibility of an intercurrent cirrhosis of the liver must be borne in mind.

In a certain proportion of cases of cancer of the stomach portions of the growth enter the general peritoneal cavity, and gravitating downward, find points of implantation in Douglas' cul-de-sac on the anterior surface of the rectum, usually 3 or 4 inches from the anus. They may be single or multiple, or they may be fused to form an area of infiltration. Ordinarily they do not exceed the size of a hazel-nut. They are to be distinguished from primary rectal growths by their implantation on the peritoneal surface of the bowel not involving the mucous membrane. In a series of 435 cases of upper abdominal carcinoma in the Mayo clinic, 307 cases being of carcinoma of the stomach, Palmer¹ found that 28 (6 per cent.) showed secondary deposits on the rectal shelf or in the cul-de-sac of Douglas, with but 2 exceptions, abdominal fluid was clinically questionable or absent.

As a rule these pelvic deposits give rise to no symptoms. They may occur with latent carcinoma of the stomach, and their detection in doubtful cases may be of the greatest help in diagnosis, hence, a routine examination of the rectum should never be neglected. Not only may the detection of pelvic growths be of diagnostic value but they may be the earliest clinical evidence of inoperability as far as a

¹ Surgery, Gynecology, and Obstetrics, February, 1910, p. 154.

radical operation is concerned. It is interesting to note that in women metastasis of the ovary may occur.

FIG. 43



Metastatic tumor in the rectal wall.

Infiltration of the umbilical ring occurs in a fair proportion of cases. It is easily detected by palpation, is of considerable diagnostic value, and, moreover, indicates inoperable extension of the disease.

PHYSICAL SIGNS AND X-RAY EXAMINATION

Physical Signs.—Inspection.—Inspection gives information more or less suggestive in a little over half of the cases that are fairly advanced. As would be expected, but little information is to be derived from this method of examination in the operable stage of early cases in which we are most anxious to establish our diagnosis. For inspection to be of any service, the light must be good, preferably oblique, and the abdomen should be sufficiently exposed.

(a) By inspection there may be noticed a fulness under the left costal arch, which does not seem to be the result of body asymmetry.

(b) In other cases a localized fulness or prominence may be seen in the epigastrium which usually shows respiratory excursions. This prominence may be due to the growth itself or it may be caused by a distended stomach, the result of pyloric stenosis.

(c) Visible evidences of tumor occurred in 42 per cent. of Osler's cases. The writer has incomplete records of the number of his cases in which the mass was discernible as growths of such size that their outline is visible, are more accurately detected by palpation.

(d) Restricted respiratory movements of the lower thorax and upper abdomen may be noted in cases of extensive adhesions.

(e) The most important evidence afforded by inspection is the detection of visible peristaltic waves in the region of the stomach passing from left to right. In every suspected case examination for signs of increased peristalsis should be made, with a strong oblique light, and a sufficient time should be allowed for the inspection, as visible peristalsis may appear only after considerable intervals of time. The examination should be made one to three hours after meals when the stomach is actively engaged in expelling its contents. Peristalsis was visible in but 4 per cent. of the writer's cases, differing materially from the experience of Osler, for in this writer's series vermicular motions of the stomach wall or localized portions of it were present in a little over 30 per cent. of the cases.

(f) A scaphoid abdomen is frequently observed in advanced cases with involvement of the peritoneum. In cachectic patients it may be a phenomenon of great diagnostic value. Visible retraction was noted in 24 out of 160 hospital cases observed by the writer (15 per cent.). Of these a palpable tumor was present in but 12, showing that marked retraction of the epigastrium usually interferes with palpation of any existing mass. If an accurate examination be demanded, the patient should be further examined in a hot bath or during light chloroform anesthesia.

(g) Symmetrical enlargement of the abdomen from accumulations of fluid was noticeable in 7 per cent. of the writer's patients.

Palpation.—(a) The chief physical sign is the presence of a palpable tumor which can be demonstrated to be of gastric origin. Unfortunately not every cancer of the stomach, even if it be of considerable size, is accessible to the palpating finger. Owing to the fact that only the lower half of the pyloric and central thirds the stomach come normally into contact with the abdominal wall, growths only in these situations are palpable from their infancy. Tumors of the cardia, of the fundus, or of the posterior wall can rarely be felt until they have attained a considerable size, or unless by reason of their situation they have caused a downward displacement of the stomach. Tumors of the pylorus and lesser curvature which may have contracted adhesions to the under surface of the liver are apt to escape detection.

In consequence the tumor is actually palpable in a comparatively small number of cases. Fenwick found a tumor in but 69 per cent. of

his cases, Osler in 76 per cent. In the writer's series a palpable tumor was found in but 54 per cent. of the patients, these figures being practically identical with those of Graham and Guthrie in the Mayo clinic, who report palpable growths in 53 per cent. of their cases.

It is often more difficult to find a tumor at certain times than at others, either because of variations in the tactus eruditus of the examiner or because of varied conditions of the fulness of the stomach by food or by gas. It thus often happens that a tumor palpable one day by one examiner will elude detection shortly afterward by another equally skilled diagnostician. Eichhorst, experienced diagnostician as he is, makes the statement that he has often treated a patient with cancer in whom a tumor was distinctly palpable, after which there might pass weeks or even months during which the most careful examination yielded negative results, so that he would often have doubted his diagnosis had it not been verified when the case came to postmortem. There may be a transmitted pulsation from the abdominal aorta, so that suspicion of aneurysm may arise. Gas may be felt gurgling through the tumor should the growth be at the pylorus, or a well-marked "pyloric squirt" may be audible through the stethoscope over the mass.

An interesting table is given by Fenwick showing the relative frequency of the difficulties encountered in detecting an existing growth.

DIFFICULTIES IN FINDING THE GROWTH IN 50 CASES

Fluid in the abdomen	38 per cent.
Tumor deeply seated, cardia, fundus, posterior wall	30 per cent.
Tumor small	20 per cent.
Excessive tenderness	12 per cent.
	<hr/> 100 per cent.

Situation.—Tumors of the stomach, especially those situated in the cardiac end, may be found in many portions of the abdomen, even in the pelvis or one or the other iliac fossa. The ordinary situation, however, is shown in the following table from figures given by Osler and Fenwick and from the writer's series.

	Fenwick. Per cent.	Osler. Per cent.	Lockwood. Per cent.
Umbilical region	37	22	38
Epigastrium	28	41	25
Right hypochondrium	17	15	19
Left hypochondrium	16	16	18
Hypogastrium	2		
Left costal arch		6	

Size.—The tumor may vary in size from a small, barely palpable lump the size of a horse-chestnut to a large mass filling the entire epigastrium. The smallest tumors encountered are usually those of the pylorus, anterior surface, or lower curvature, in the situations where the growth is apt to come in direct contact with the abdominal wall. Enormous masses may be encountered when the neoplasm has involved the greater part of the stomach, or has invaded neighboring parts by direct contiguity.

Shape.—The shape may be oval, rounded, tubular, or irregular. A general infiltration of the wall of the stomach gives rise to a mass which in shape resembles that of the normal organ, the lower margin of which is more distinct in outline than the upper. The tumor formed by diffused infiltrated cancer causing a contraction of the stomach, the so-called "water-bottle" type, may be felt as a narrow firm cord, resembling a sausage in shape, extending from below the left costal arch. It may be mistaken for the thickened puckered omentum of chronic peritonitis, tumors of the colon, or the lower edge of a diseased liver. Tumors of the fundus often attain considerable size and projecting beyond the left costal arch may so closely resemble enlargements of the spleen that a differential diagnosis by physical examination alone cannot be made. Three such cases have lately come under the writer's observation.

In a case reported by Jellett,¹ of a woman who complained only of pain and swelling of the abdomen, a semicystic tumor could be felt about the size of a seven months' pregnancy which turned out to be a malignant tumor originating in the stomach.

Density.—The growth if small may be smooth and give the feeling only of an indefinite solid body. The majority of cancers, however, are irregular in shape, show slight nodosities on the surface, and are of a characteristic stony hardness.

Motility.—Tumors of the stomach almost always exhibit a certain degree of motility, depending on their situation and upon the presence or absence of limiting adhesions. Tumors of the pylorus are usually freely movable unless the excursion of the growth is limited by adhesions to parts that are fixed. Not all adhesions limit the motility of the neoplasm, for in 13 cases reported by Osler in which the mass moved freely with respiration and palpation, adhesions were present in 9.

Changes in the position of the mass may be due:

1. To changes in the size and position of the stomach during digestion, or as the result of inflation.
2. To the effect of respiratory movements.
3. To the mechanical effect of palpation.

¹ British Med. Jour., March 23, 1912.

1. Tumors are often dragged down when the stomach is full and move up when the stomach empties itself again. The excursions during the various stages of digestion may be quite remarkable.

Inflation of the stomach with gas, either as a result of flatulence from natural causes or by the artificial inflation by gas as a method of examination, usually produces characteristic changes in the position of the growth. By inflation pyloric cancers usually move to the right and downward, rarely to the left and upward. Tumors of the lesser curvature tend to disappear, because by inflation the stomach revolves on its longitudinal axis so that the upper curvature looks more directly backward and the lower curvature looks more directly forward. Tumors of the greater curvature thus become more evident. If adherent to the pancreas no change in the position of the mass is apt to occur with inflation. It need hardly be emphasized that the inflation of the stomach with gas for diagnostic purposes should be done most cautiously to avoid perforation.

2. *Respiratory movement* is almost regularly present unless the tumor be adherent to the abdominal wall or pancreas. In the writer's cases 42 per cent. were freely movable on respiration or by palpation, 12 per cent. were fixed, while no mention was made of motility in 46 per cent. In Osler's patients motility with respiration or palpation occurred in 60 per cent. According to Boas, tumors of the pylorus do not readily move with inspiration unless they are adherent to the liver, whereas, cancer of the curvatures show, as a rule, well-marked respiratory displacement. With this statement the writer cannot agree, as in his experience an equal motility in these two situations has been observed. It is often difficult to distinguish between a respiratory gliding of the abdominal wall over a tumor and actual respiratory movement of the tumor itself.

3. Motility during palpation is usually evident to the examining hand, and some idea as to the location of the growths and the presence or absence of adhesions may be deduced from the extent of the excursions of the mass.

In a certain number of cases expiratory fixation may be observed. This physical sign consists in the grasping of the tumor during the period of deepest inspiration, and preventing its ascent during expiration unless the grasp be released. Expiratory fixation indicates usually the absence of adhesions, especially to the liver, but the converse does not always hold true. The absence of expiratory fixation does not necessarily prove the presence of adhesions.

Tenderness to some extent can almost invariably be elicited by deep palpation over the diseased area, or of the mass itself. The tenderness is, however, seldom extreme, much less so than in ulcer, is more diffuse

than in the latter disease, and is but rarely accompanied by the cutaneous hyperesthesia and the dorsal point of tenderness that so often occur with gastric ulceration.

In many cases palpation yields only a localized sense of resistance, especially in the early cases before the growth is large enough to be distinctly palpable. Growths of the posterior wall, or those in greater part covered by the liver or costal arch, may give only an indefinite sense of resistance even though they be actually of considerable size.

Rigidity was appreciable in 18 out of 170 cases in the writer's series in which mention is made of its presence or absence (11 per cent.). Of these 18 cases a tumor was palpable in 5, and undetected in 13, showing how easily rigidity of the abdominal wall may interfere with other methods of examination. Rigidity may be localized in the epigastrium or over the head of either rectus muscle, or there may be general abdominal rigidity, often associated with a scaphoid appearance of the abdomen.

Rigidity indicates peritoneal involvement, and while not especially diagnostic of cancer, it is a sign of great importance, as it implies an extension beyond the actual confines of the stomach so as to render any hope for radical relief by operation highly improbable.

Palpation may also detect hypertonus of the stomach in cases of pyloric implantation. With the hand gently pressing downward upon the epigastrium there may be felt from time to time a sense of stiffening of the stomach wall, so that the organ can be quite readily mapped out. The duration of the period of stiffening is usually quite short. If the phenomenon be more pronounced than this there may be felt a marked prominence of the region of the fundus, and at the acme of the contraction the patient may experience considerable discomfort, or even pain. With extreme pyloric contraction there may be peristaltic waves passing from left to right which are distinctly evident to the examining hand. The march of the peristalsis is quite stately, and occupies an appreciable period of time.

By palpation we may make out enlarged supraclavicular glands on one or both sides of the neck, infiltration of the umbilical pit, or the presence of metastases in the liver causing hard irregularity of its contour or lower edge. Rectal examination for the detection of possible implantation in the vesicorectal pouch should never be neglected.

Gastric Dilatation with Cancer.—There is considerable difference of opinion as to the frequency with which dilatation of the stomach accompanies the pyloric stenosis of malignant origin. Osler thinks that dilatation is relatively more commonly due to cancer than to any other cause, as in the same period of time he found that of 67 cases of dilatation of the stomach 42 were due to cancer. In his series

of 150 cases of cancer dilatation either before or after inflation was recognizable in 42 instances (28 per cent.).

On the other hand, Boas and Broadbent claim that gastric dilatation is not the rule. The writer regrets that his records are not complete enough to give statistical proof one way or another, but is strongly inclined to the belief that while gastric dilatation may occur, it is usually to a very slight degree, and that dilatation, to the extent that we see it with benign stenosis, is comparatively rare except in instances of carcinomatous degeneration of callous ulcer of the pylorus.

X-ray Examination in Cancer.—1. The *x*-ray examination of cancer of the stomach, while usually convincing in cases of advanced growth, may be totally inconclusive in its early stages. In some of the early cases, however, our suspicions may be aroused by certain deviations from the normal in size, shape, and density of the stomach, by distortion or by lack of motility.

In some of the early stages the picture may resemble that of ulcer. Bismuth residue after the expiration of six hours indicates atony or some form of pyloric narrowing, either spasmodic or organic, and the radiographs of ulcer near the pylorus, of cancer in that vicinity, and of pylorospasm accompanying gall-bladder or appendicular disease, may be so similar that a differential diagnosis by this form of examination alone is quite impossible.

In ulcer and cancer we may have the same drawing of the pylorus upward and to the left, forming the "snail-like" contour described in ulcer. All we can say is that there seems to be an organic pyloric lesion present, the exact nature of which can only be determined by the clinical history, physical examination, gastric analyses, and the clinical course of the ailment, and possibly even then operation alone can decide. The evidence of adhesions may be present in both conditions (see *x*-ray of ulcer). According to Holzkecht, a diagnosis of early carcinoma can be made in a patient with achylia, if, six hours after the meal, bismuth residue is found in the stomach, provided that the head of the bismuth column has at this time reached the splenic flexure, and that the second bismuth meal shows a normal stomach shadow. Holzkecht's reasoning is that as normally the head of the bismuth column should in six hours reach only the hepatic flexure, we are dealing with hypermotility when the splenic flexure is in bismuth shadow, which, when the pylorus is free, is a regular accompaniment of achylia. The bismuth residue, moreover, demonstrates an achylia with stagnation. The stipulation that the contour of the stomach after the second bismuth meal should be normal, would exclude atony and many of the cases of ulcer.

It would seem to the writer, however, that this view of Holzkecht's

is too extreme, as ulcers near the pylorus which occasionally happen to be associated with achylia, may produce the same radiographic picture, and that, furthermore, a differential diagnosis from pylorospasm due to gall-bladder disease, accompanied as so often is the case, by achylia, would be quite impossible. Morphinism must always be excluded as a possible cause, as in this addiction pylorospasm and bismuth retention may be found in the stomach, which, after the second bismuth meal, is seen to retain its normal size and shape, but in morphinism there would be no bismuth at splenic flexure.

2. When cancer is so extensive and infiltrating the radiograph may show nodular indentations "similar to finger prints" (Cole), where the bismuth shadow is not as intense as elsewhere, or does not appear at all.

The rugæ in the infiltrated area are absent.

When the destructive process is extensive, large areas of the organ may be entirely obliterated, the ragged edge of the uninvolved area sharply limiting the outline of the bismuth shadow.

Should the pylorus be obstructed by the neoplasm, the narrow constricted lumen of the pylorus may give passage to an abnormally thin line of bismuth, which is sinuous and contorted in outline. Sometimes the bismuth shadow of the pyloric end of the stomach narrows down gradually in the form of a cone, with a small outlet at the apex.

In other instances a thread-like shadow may issue directly from the sharply defined edge of an apparently normal portion of the pyloric extremity of the stomach.

Peristaltic contractions become less active upon entering the conical area.

In some instances the stomach is found to be empty in six hours, but has lost its normal hook shape, and has assumed a "horn shape," being short and placed diagonally. It is impossible, however, by the radiographic plate alone to determine whether this horizontal, contracted stomach is due to hypertonicity or to infiltration of its wall by cicatricial tissue or by scirrhus.

In cases of inoperable diffuse carcinoma of the entire stomach, including the cardia, we are apt to find the stomach empty in six hours, the head of the bismuth column at or beyond the splenic flexure, a bismuth deposit in the lower esophagus after six hours. The second bismuth meal shows us a greatly shortened distorted organ lying obliquely in the abdomen, and the bismuth immediately after ingestion beginning to flow freely out of the stomach, indicating insufficiency at the pylorus. The radiographic evidence of pyloric stenosis will be described under the heading of pyloric stenosis.

PLATE IV

Fig. 1



Fig. 2

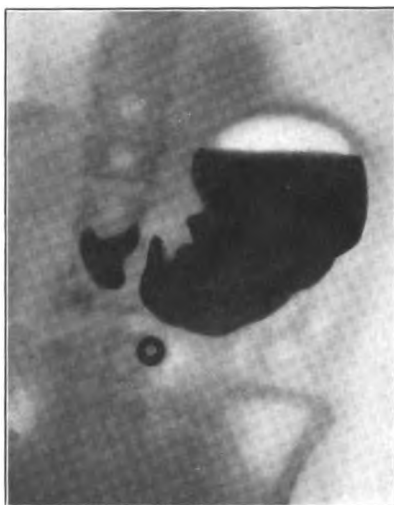
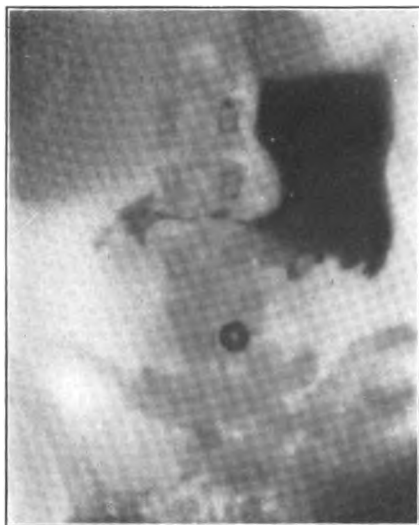


Fig. 1.—Carcinoma of Body of Stomach. Rigid Patency of Pylorus. Increased Motility of the Colon, Head of Bismuth Column in Six Hours being in the Descending Colon. Fluoroscopic examination of this case shows duodenum filled with bismuth as far as the duodenojejunal angle within ten minutes after the ingestion of the bismuth meal. (Radiologist, Dr. Leaming.)

Fig. 2.—Carcinoma of the Lesser Curvature, not Involving the Pylorus. (Radiologist, Dr. Le Wald.)

Fig. 3



Carcinoma of Pyloric Half of the Stomach. (Radiologist, Dr. Leaming.)

Fig. 4



Carcinoma of the Pars Media.

PLATE V

Fig. 1

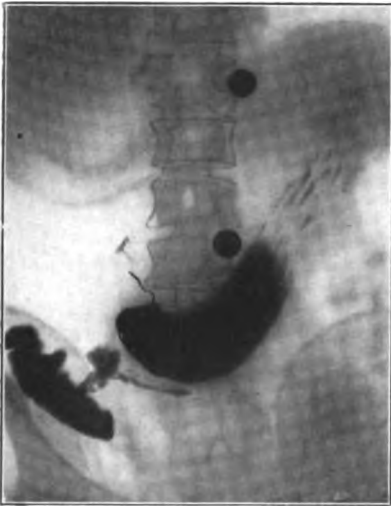


Fig. 2



Fig. 1.—Cancer of the Pylorus Producing Stenosis. Noteworthy is the undershot line of the greater curvature. (Radiologist, Dr. Busby.)

Fig. 2.—Carcinoma of Pylorus, resembling Benign Stenosis. Stomach dilated and displaced. (Radiologist, Dr. Le Wald.)

Fig. 3



Fig. 4



Fig. 3.—Carcinoma of the Pyloric End of Stomach, producing Stenosis. (Radiologist, Dr. Le Wald.)

Fig. 4.—Carcinoma of Pylorus, producing Tight Stenosis. (Radiologist, Dr. Leaming.)

PLATE VI

Fig. 1

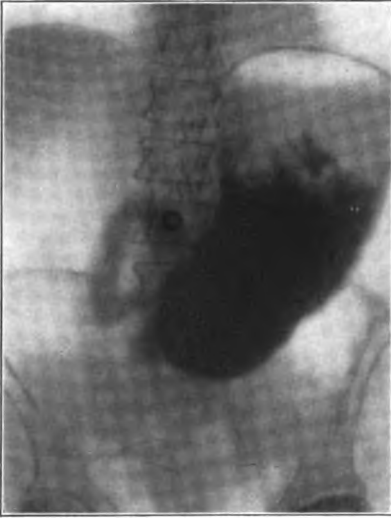


Fig. 2



Fig. 1.—Carcinoma of the Lesser Curvature, with Insufficiency of the Pylorus. (Radiologist, Dr. Le Wald.)

Fig. 2.—Diffuse Carcinoma of the Entire Stomach, with Insufficiency of the Pylorus and Hypermotility of the Colon. (Case of Dr. J. W. Weinstein; radiologist, Dr. Leaming.)

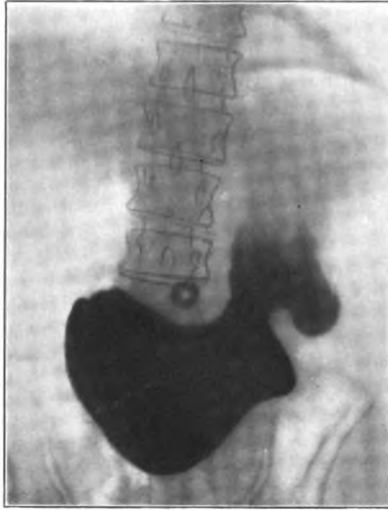
Fig. 3



Carcinoma of the Lesser Curvature. Deep incisure of greater curvature, resembling the incisure commonly seen with ulcer in this situation. (Radiologist, Dr. Le Wald.)

PLATE VII

Fig. 1



Carcinoma of Lesser Curvature. Deep Broad Incisure in Greater Curvature. The incisure in these cases is apt to be broader than is the case with ulcer. Radiologist, Dr. Le Wald.)

Fig. 2



Ulcer of Lesser Curvature, causing Spasm and Drawing Up of Greater Curvature. (Radiologist, Dr. Busby.)

In some instances of cancer of the lesser curvature there may be the same deep fixed incisure as is seen in ulcer. In other cases carcinoma of the greater curvature will produce the same appearance.

Gastric Analysis.—It is of the utmost importance to examine the gastric contents in every suspected case of cancer, not only to determine if possible the presence of the growth, but also its situation with reference to a possible operation. Every suspected case should be examined, not only once but repeatedly, unless the passage of a tube be contraindicated by extreme physical weakness, or by recent and severe hemorrhage.

Examinations of test breakfast are commonly made, but the conditions of the fasting stomach have generally been neglected. The writer believes that in cancer, examination of the fasting stomach gives as important testimony of the presence of a neoplasm as examination of the test breakfast, and therefore should never be omitted.

The writer's procedure in examining a suspected case is as follows:

The patient eats during the day his accustomed meals. Between 10 and 11 o'clock in the evening he is given a meat sandwich, preferably of cold roast beef, although any meat will serve the purpose, and a glass of water. Thereafter nothing is to be given, not even water, until the following morning at 8 to 9 o'clock, at which time a stomach-tube connected with an aspirating bulb is passed and the contents of the stomach are withdrawn. Unless the presence of appreciable quantity of fresh blood in the gastric contents or extreme physical weakness should contra-indicate further examination, the patient is to be then given a roll, without butter, and a glass of water. One hour after beginning such a test meal the contents of the stomach are again withdrawn. If food-stasis is suspected, a tablespoonful of raisins or dried currants is given with the evening sandwich. For those who find the early morning hour inconvenient, as often is the case with those who live out of town and who would therefore be compelled to travel on an empty stomach, the following modification is permissible:

A breakfast of a small portion of steak, a portion of boiled rice without sugar or milk or butter, and a breakfast roll are to be given at 7.30 A.M. and the contents withdrawn four hours later, after which the test breakfast is to be given as before.

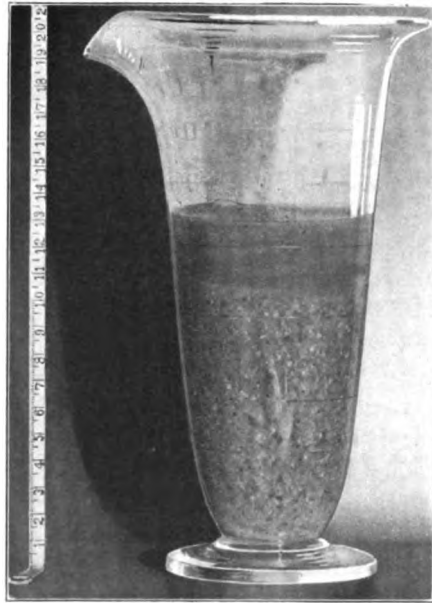
In case only one examination by the tube is permitted, the steak, rice, and bread breakfast may be given at 7.30, the test breakfast given at 12.30, and the whole contents removed one hour later. A rough estimate of gastric motility is afforded by such an examination, though by no means as exact as by either of the preceding methods.

The writer has had no occasion to use the lactic and free oatmeal supper recommended by Boas.

By such a double examination of gastric contents, the writer has obtained importance evidence of disease in 85 per cent. of the cases.

Examination of the Fasting Stomach.—Fasting Stomach Normal.—In 30 per cent. of the patients examined, the fasting stomach showed absolutely normal conditions, the proportion of such normal fasting examinations being far greater in private than in hospital practice. The earlier the patient comes under observation the more apt is the fasting examination to approach the normal. The largest number of those with normal fasting conditions were found among the cases of cancer that did not involve the patency of the pyloric orifice. It is interesting to note that in about one in ten of these normal fasting cases, examination of the test breakfast gave positive indications of organic lesion in the stomach. This fact alone proves the importance of the double test.

FIG. 44



Fasting contents of gastric carcinoma involving the pylorus.

Evidences of Motor Error in Fasting Stomach.—Seventy per cent. of all patients with cancer of the stomach present evidences of grave motor error by the examination of the fasting stomach. Such evidences are more commonly seen in hospital than in private practice. Seventy-eight per cent. of the writer's hospital cases gave evidences of food stagnation, whereas in only 61 per cent. of the private cases could this evidence be obtained.

The appearance and quality of the contents of the fasting stomach indicative of stasis, differ considerably in the different cases. In some instances there are aspirated small quantities only of clear fluid, containing food remains in minute though in appreciable amount. The quantity of fluid varies from 15 c.c. to large and copious amounts, often exceeding a liter, the quantity giving a fair estimate of the degree of pyloric narrowing. The fluid may be discolored by food remains or by altered blood. The aspirated contents may be clear, tinged with a very slight sedimentary layer, or may consist largely of gross and obvious food remains. The odor may be offensive in the extreme in advanced cases in which the growth is ulcerating.

Chemical Examination.—The chemical examination differs in different cases.

1. In 18 per cent. of cases the fluid resembles that of ordinary hypersecretion in that it is clear and is inoffensive in odor. Food remains are present in varying amount, but the chief characteristics are those of benign hypersecretion. The quantity has varied in the writer's case from 50 c.c. to 500 c.c. Total acidity ranges between 40 and 120, the acidity being due entirely to free and combined hydrochloric acid. In this form lactic acid and the Oppler-Boas bacilli are absent. Sarcinæ may be occasionally found. Blood is present in small but appreciable quantities in one-half the cases.

All the cancer patients in the writer's experience who show this form of fasting secretion are instances of growth at the pylorus. A previous ulcer history is uncommon, nor in the cases followed to autopsy were there any obvious traces of former ulceration.

2. In other cases the gastric contents in the fasting state contain lactic acid. Thirty-six per cent. of the patients examined by the writer gave this reaction. The quantity of aspirated contents varied usually from 15 c.c. to 75 c.c., but occasionally very large quantities could be withdrawn, in several instances exceeding 2 liters. Food remains are constantly present, and are ordinarily offensive. The total acidity ranged from 30 to 80, occasionally higher than this. Lactic acid is distinctly present. Oppler-Boas bacilli are easily demonstrated. In one-quarter of the cases giving lactic acid reactions in the fasting state free hydrochloric acid was also present, and when both hydrochloric acid and lactic acid occur together, the total acidity is usually high, in one instance being 182. Such an analysis would seem to indicate a chronic ulcer at or near the pylorus which has undergone carcinomatous changes.

In three-fourth of the cases with lactic acid in the fasting contents, hydrochloric acid is absent. The contents vary from 45 to 450 c.c., food remains are constantly present in varying amounts and are fre-

quently offensive. Gastric contents giving these reactions have been found in a little over 25 per cent. of the writer's cases.

3. There is a group of cases comprising 16 per cent. of the writer's series in which there are evidences of food-stasis but in which the total acidity is so slight as to be negligible. Both lactic acid and hydrochloric acid are absent, although in many instances the Oppler-Boas bacilli are found. Blood is almost regularly present, often in sufficient quantities to neutralize the small degree of acidity that otherwise would exist. The quantity of contents has varied in the writer's experience from 30 c.c. to 1000 c.c. and is largely composed of food remains, usually extremely foul, occasionally even fetid. Undigested meat fibers are almost regularly present. These characteristics of the fasting contents afford conclusive proof of advanced malignant disease, and occur with special frequency in hospital cases.

To recapitulate the results of the fasting stomach examination in the writer's series (224 cases)

Normal	30 per cent.
Stagnation (a) With hydrochloric acid alone	18 per cent.
(b) With lactic acid	27 per cent.
(c) With both lactic and hydrochloric acid	9 per cent.
(d) Without either lactic or hydrochloric acid	16 per cent.
	<hr/> 100 per cent.

Of the cases in which fasting contents were obtained (70 per cent. of the total number):

Hydrochloric acid was present alone	25.5 per cent.
Hydrochloric acid was present with lactic	12.9 per cent.
Total percentage containing free hydrochloric acid	<hr/> 38.4 per cent.
Lactic acid was present alone	38.67 per cent.
Lactic acid was present with hydrochloric acid	12.90 per cent.
Total percentage containing lactic acid	<hr/> 51.57 per cent.

Trichomonas, *megalostoma* and various forms of flagellated infusoria may occasionally be found in the fasting stomach. They occur only in alkaline or neutral media, and are more common in cases of carcinoma of the cardia or fundus without stagnation.

Cohnheim who has drawn attention to this subject regards them as suggestive of early cancer, and with him various other authors agree. The infusoria are, however, often present in the tartar of carious teeth and may readily be swallowed. They may be found in achylia of non-cancerous origin, and are therefore not pathognomonic of malignancy.

Their presence in the stomach implies merely the combination of carious teeth and a lack of acid secretion in the fasting stomach.

Pus cells commonly occur in the gastric contents of advanced cases. They may arise either from the ulceration of the growth or from a localized phlegmon of the adjacent portions of the wall of the stomach. To be of any diagnostic significance purulent inflammation of other parts, such as the mouth, gums, tonsils, throat and bronchi, must be excluded. Pus that is discernible by the naked eye usually indicates adhesions to and perforation into an adjacent solid viscus forming an abscess cavity communicating with the stomach.

Fragments of cancer tissue may be found in the fasting contents, but they are rarely of use for early diagnosis. In many instances the fragments are necrotic and are unfit for histological examination.

Examination of the Test Breakfast.—It is a general rule that if evidences of stagnation are found in the fasting state they will also be present in the test breakfast. If the fasting stomach, however, be emptied of its contents just before the giving of the test breakfast, or if the patient has recently vomited, the test breakfast will be nearer the normal than if the stomach had not thus been emptied before the test breakfast was taken. At first sight it may even be that the test breakfast shows very little evidence of impaired motility, but on more careful examinations there is almost always found conclusive evidence of stasis in the test breakfast if stagnation has been also evident in the fasting state. Such apparent discrepancies between the fasting examination and the test breakfast is not at all uncommon. The following case is illustrative:

N. B., aged fifty-nine years, was well and of good digestion until ten months ago, when he began to lose flesh and strength, and food became repulsive to him. Seven months ago a pain began in the region of the stomach, of a dull aching character, usually occurring about an hour after eating and only partially relieved by vomiting. For three months he has vomited nearly every evening a large quantity of offensive material.

Physical Examination.—A mass of the size of a mandarin is distinctly palpable in the epigastrium, hard, irregular, and slightly tender, descending with respiration and capable of expiratory fixation. Patient shows marked cachexia. Red cells, 2,600,000; hemoglobin, 21 per cent.

Examination of fasting stomach shows 520 c.c. of brownish offensive contents separating on standing into two layers, the upper three-fifths being of liquid, the lower two-fifths being composed of food remains. Total acidity 70, free hydrochloric acid 56. Blood positive. No lactic acid, sarcinæ or Oppler-Boas bacilli present.

Test breakfast given immediately after the removal of the fasting contents shows 90 c.c. of well-digested food remains of normal acidity. Although to the eye the test breakfast appeared perfectly normal, microscopical examination showed the presence of many meat fibers and other food remains sufficient to afford conclusive proof of stagnation.

Normal Test Breakfast.—The test breakfast in gastric cancer was normal in 15 per cent. of the writer's cases.

While it is a general rule that normal fasting conditions and normal test breakfasts go together in the same patient, it may happen that one or the other may show a deviation from the normal sufficient to afford some clue to the diagnosis, hence, the writer's insistence upon the double examination, the importance of which is shown in the following instances:

Fasting stomach normal. Test breakfast indicative of cancer.

J. C., aged fifty-seven years, was practically well until seven weeks ago, when he began to complain of weakness, loss of appetite, and shortness of breath. He became so weak that he was obliged to stay in bed nearly all the time. He has had no dyspeptic symptoms whatever.

Physical Examination.—A firm, slightly tender mass is palpable in the epigastrium just to the left of the median line, descending with respiration. Signs of fluid are present at both bases behind.

Examination of the fasting stomach shows the organ to be absolutely empty.

Test breakfast: 15 c.c. well digested breadstuffs without free or combined hydrochloric acid. Lactic acid and Oppler-Boas bacilli present. Blood positive. Autopsy showed carcinoma of the lower curvature with metastases in the liver, both lungs and pleuræ and pericardium.

Case with stasis in the fasting stomach. Test breakfast normal.

B. B., aged thirty-eight years, no previous gastric history. Seven months ago she began to raise gas of an offensive odor and to suffer from occasional attacks of diarrhea. She has not lost flesh nor strength but comes for advice because she has herself been able to feel a lump in her stomach.

Physical Examination.—A freely movable mass of the size of a lemon is palpable in the pyloric region.

Examination of the fasting stomach: 120 c.c. of well-digested food remains separating on standing into two layers of equal depth. Total acidity 50, free hydrochloric acid 28. Lactic acid, Oppler-Boas bacilli, and minute traces of blood are present.

Test breakfast: 200 c.c. well-digested breadstuffs of normal appearance. Total acidity 64, free hydrochloric acid 30, no trace of lactic acid, no Oppler-Boas bacilli or blood.

Abnormality of Test Breakfast.—An abnormality in test breakfast occurred in 85 per cent. of the patients examined. It must be borne in mind, however, that many of these patients were first seen in the advanced stages of the disease, long past the operative period. In those who are seen earlier in the course of their malady the proportion of abnormal test breakfast is naturally much less. It is more than probable that the majority of early cancers in their operative stage give gastric analysis which deviate so slightly from the normal that it is not possible to make any diagnostic deductions from them whatever. This is unfortunate, because if we wait for positive evidence of malignancy we wait too long.

Abnormalities in the test breakfast are of three varieties:

(a) In 52 per cent. of the cases lactic acid was present while hydrochloric acid was absent both in the free and combined forms.

The quantity of test breakfast expressed varies from a few c.c. up to the larger quantities indicative of pyloric stenosis. The total acidity ranges from 20 to 110 or even higher, but is almost regularly less than in the contents obtained in the fasting state. The odor may or may not be offensive. *Sarcinæ* may very occasionally be found, their presence suggesting the existence of a previous ulcer. *Oppler-Boas* bacilli are almost regularly present and are especially abundant in the minute brown coagula that are found in the test breakfast in many instances.

Blood reaction is almost regularly positive. Although this form of test breakfast has been considered almost pathognomonic of cancer it may occur in instances in which no neoplasm or other organic disease can be detected by exploration. Such cases, while uncommon, do occur, and while in some cases no adequate cause for such an analysis can be adduced, in other instances definite lesions other than cancer appear. Syphilitic pyloric lesions and occasionally the sclerosing form of linitis plastica may be accompanied by the same chemical type of test breakfast.

(b) In 21 per cent. of the writer's cases neither hydrochloric acid nor lactic acid was present. The appearance of this form of test breakfast varies considerably. In some instances the appearance is apparently normal, the breadstuffs being well digested, of normal consistency and quantity, and though chemical examinations show merely an absence of hydrochloric acid. In other cases the test breakfast is poorly digested, contains gastric mucus in fairly large quantities, resembling exactly in appearance the test breakfast obtained in cases of an acid catarrhal gastritis. In only one instance in the writer's cases was there present the dryish, squeezed-out appearance so characteristic of dry achylia.

Although these findings are not at all characteristic of cancer, as they occur frequently enough in non-malignant achylia and in sub-

acid gastritis, they are suspicious if they occur in a patient of adult years who has been losing flesh and strength without assignable cause. Repeated examinations should be made in the hope of finding more definite indications of malignancy at some subsequent examination. It is interesting to note that of the patients of this type who came under the author's observation, a clinical history strongly suggestive of malignant disease was found in every instance, and, moreover, that in 82 per cent. of these patients a palpable growth was found in the epigastrium, so that a diagnosis could be made without the help afforded by gastric analysis. In many instances corroborative signs were found in the examination of the fasting stomach. It should be remembered, however, that in the author's series of cases are naturally included only those in which the diagnosis of cancer had been positively made.

(c) Both hydrochloric acid and lactic acid were present in the test breakfast of 12 per cent. of the cases examined. In all these instances pyloric obstruction was present, and a previous gastric history suggested an ulcer was present in one-half of these. The total acidity varies usually between 50 and 80, free hydrochloric is present from 20 to 60. Lactic acid reactions are regularly strongly positive in the majority of these cases. Hypersecretion giving reaction for free hydrochloric acid was present in the fasting state as well, and the patients frequently vomit large quantities of brown acid fluid.

To tabulate the number of cases in which hydrochloric acid and lactic acid were present, the following figures are reduced:

Hydrochloric acid was present alone	15 per cent.
Hydrochloric acid was present with lactic acid	12 per cent.
Total number with free hydrochloric acid	27 per cent.
Lactic acid was present alone	52 per cent.
Lactic acid was present with hydrochloric acid	12 per cent.
Total number of cases with lactic acid present	64 per cent.

These figures are quite different from those given by Graham and Guthrie in the Mayo clinic, for these writers found hydrochloric acid present in 46.7 per cent. (70 out of 150 cases), while lactic acid was present in 42.7 per cent. (64 out of 150 cases). This is to be explained by the fact that in the Mayo clinic there is a larger number of early cases positively determined by exploration, whereas, medical statistics embrace more cases in advanced stages of malignancy, in whom the changes in the test breakfast indicative of carcinoma are marked.

Specific Tests Proposed for the Diagnosis of Gastric Cancer.—The prognosis in gastric cancer depends so directly upon an early

diagnosis that the question of developing a reliable test which shall differentiate the early cases of this disease from those conditions which closely resemble it clinically, remains today one of the great unsolved problems in the field of medical research. Many tests have been proposed for this purpose.

Serological Tests.—These may be classified according as they depend upon (1) complement deviation, (2) meiotagmin, and (3) isohemolysis reactions.

COMPLEMENT DEVIATION REACTIONS.—*Normal Serum Hemolysis.*—That change which occurs in red blood corpuscles when their hemoglobin is set free and passes into solution is called *hemolysis*.

It may be produced by contact with many substances, among which is blood serum. The power of blood serum to produce hemolysis is dependent upon the coördinate action of two distinct elements contained therein, which are known as *amboceptor* and *complement*.

Amboceptor is absorbed by the red cells and renders them susceptible to the dissolving action of complement. Thus amboceptor is the sensitizing, and complement the dissolving agent. Amboceptor alone has no power to hemolyze red cells. Complement has the power of hemolysis of red cells only when they have been sensitized by amboceptor.

Amboceptors active against the red cells of certain species exist naturally in normal sera. Also by repeated injection into an animal of sheep's red cells, there is developed in the serum of that animal an amboceptor active against sheep's cells. Thus there are two kinds of amboceptors which are known as *normal* and *immune* amboceptors respectively. Immune amboceptors are the more dependable and therefore are the more used. Amboceptor for use against sheep's red cells is often obtained from the serum of rabbits which have been repeatedly injected with sheep's red cells.

Amboceptor and complement in a serum may be separated from each other because of different physical characteristics. Amboceptor is the more stable, retains its activity for a long time, and is not injured by exposure to a temperature of 55° C. Complement deteriorates rapidly, and is destroyed by exposure to this temperature for thirty minutes. For practical purposes complement is obtained usually from the serum of freshly drawn guinea-pig's blood.

If, under certain conditions, to a suspension containing a certain amount of washed sheep's red cells there be added proper amounts of amboceptor (obtained as described above) and complement (fresh serum from a guinea-pig) the red cells will be hemolyzed. This phenomenon is known as normal serum hemolysis. If either amboceptor or complement are missing hemolysis will not take place.

Antigens and Antibodies.—Various substances when injected into animals cause the formation of corresponding specific reaction products in those animals. Substances which have this power are called *antigens* and the reaction products are called *antibodies*. Bacteria and certain forms of foreign protein when injected into animals are antigens because they give rise to corresponding antibodies. The infectious agent in syphilis may be considered an antigen, for when it is introduced into the body it gives rise to specific syphilitic antibodies. Its cultivation to any extent has thus far been impracticable, but it has been found that extracts of certain syphilitic tissues, such as an aqueous extract of syphilitic fetal liver, act as antigen toward syphilitic antibodies.

Complement Deviation.—The relationship between antigen and antibody presents two striking characteristics: (1) A given antibody reacts, outside of the body, with the antigen from which it resulted and with no other. (2) A combination of antigen and its specific antibody, when added in proper proportions to the elements necessary for normal serum hemolysis, has the power of absorbing complement, thus preventing the hemolysis. The combination of antigen and any other than its specific antibody will not result in the absorption of complement. This phenomenon is known as *complement deviation* or *complement fixation*, and it is the basis of the Wassermann test and its modifications for the diagnosis of syphilis.

Application of Complement Deviation to Diagnosis.—The five elements in the Wassermann reaction and the concrete substances by which each may be represented are:

1. Syphilitic antigen—aqueous extract of syphilitic fetal liver.
2. Syphilitic antibody—that contained in the blood serum of a syphilitic patient.
3. Red blood corpuscles—those from sheep's blood, washed and put in a suspension of definite strength.
4. Amboceptor—obtained from the serum of a rabbit which has been repeatedly injected with sheep's red corpuscles.
5. Complement—fresh guinea-pig's blood serum.

The accurate combination of these five elements, under suitable conditions, will not result in hemolysis, because the combination of specific antibody and its antigen has absorbed complement and therefore it cannot exert its hemolyzing action on the red corpuscles sensitized by amboceptor. Such a result is a positive reaction, for the serum from the patient must have contained syphilitic antibodies.

If, however, blood serum from a non-syphilitic individual be used instead of that from a syphilitic patient, there will be no syphilitic antibodies present to combine with antigen—for the absorption of complement, and complement will remain free to produce hemolysis.

This is a negative reaction; the serum used could not have contained syphilitic antibodies.

Thus, in this case, with a known antigen, the presence or absence of its specific antibodies in an unknown serum may be demonstrated.

An attempt has been made to apply the principle of complement deviation, which has been of such remarkable value in the diagnosis of syphilis, to the diagnosis of early cancer. Many elaborate researches have been undertaken in the hope of finding an antigen—perhaps an extract of cancerous tissue—which would combine with the antibodies supposed to exist in the serum of patients suffering from cancer, to absorb complement. Aqueous and alcoholic extracts of cancerous tumors have been most tried as antigens. Among the investigators in this field are: Sampietro and Tesa,¹ Simon and Thomas,² Ravenna,³ Sisto and Jona,⁴ Weinburg and Mello,⁵ De Marchis,⁶ Guillot and Daufesne,⁷ Hirschfeld,⁸ and von Dungern.⁹ The results obtained by them are not altogether uniform. Some obtained positive reactions in cases that were not cancerous and many in syphilis. Some failed to obtain reactions of any kind; others noticed that in all cases in their series giving negative reactions were those of cancer of the uterus or alimentary tract. von Dungern's results are more nearly consistent than others. However, it must be concluded that the actions of the antigens thus far used have not been specific enough to certainly differentiate cancer from syphilis, benign newgrowths, and other conditions.

MEIOSTAGMIN REACTIONS (*μειων*—smaller, and *σταγμα*—a drop). Traube observed that the addition of a toxin to an antitoxin produced a lowering of the surface tension of the fluids, which is measured by measuring the size of the drops with a stalagmometer. Ascoli found that this phenomenon occurred when serum from patients suffering from certain diseases was mixed, after suitable dilutions, with an appropriate antigen. Ascoli and Izar¹⁰ have attempted to prepare an antigen which would give the characteristic reaction with serum

¹ Sampietro and Tesa. *Annal. d'Igiene Sperim.*, 1908, p. 657.

² Simon and Thomas. *Jour of Exp. Med.*, x, 673.

³ Ravenna. *Arch. Scien. Med.*, 1909, No. 6.

⁴ Sisto and Jona. *Clin. Med. Ital.*, 1909, xlviii, 289.

⁵ Weinburg and Mello. *Bull. Assoc. Franç. pour l'étude du cancer*, 1910.

⁶ De Marchis. *Lo Sperimentale*, 1910, p. 969.

⁷ Guillot and Daufesne. *Bull. Assoc. Franç. pour l'étude du cancer*, 1910, p. 34.

⁸ Hirschfeld. *Deutsch. med. Woch.*, 1911, No. 27.

⁹ von Dungern. *Münch. med. Woch.*, January 9, 1912.

¹⁰ Ascoli and Izar. *Münch. med. Woch.*, Nos. 8, 18, and 22

from cancer patients. Although some, with this method, have obtained a positive reaction in a small percentage of other pathological conditions, the results have thus far been very encouraging. Krauss, von Graff, and Ranzi¹ consider it the most reliable of the serum reactions.

ISOHEMOLYSIS REACTIONS.—The tests in this group depend upon that property of the serum of patients suffering from certain diseases which produces hemolysis of normal red blood corpuscles from the same species. Weil² showed that the serum of dogs suffering from lymphosarcoma hemolyzed the red blood corpuscles of normal but not of sarcomatous dogs. He and other observers have studied the same phenomenon in human cancer patients.

Judging from the results up to the present time from all observers, it is probable that the reaction occurs in less than half of all cases of cancer. Moreover, it has been found to take place in a few normal individuals and in a considerable number suffering from other pathological conditions.

Elsberg³ by injecting under the skin of cancerous patients a small amount of a 20 per cent. suspension of washed defibrinated blood, and observing the subsequent local reaction, has obtained results that seem remarkably constant. This test has been favorably reported upon by Leitch.

Tryptophan Tests depend upon the detection in the gastric contents of a product of the action of the proteolytic ferment which malignant neoplasms have been reported to contain. It is maintained that the normal gastric ferments have no power to carry the digestion of proteids farther than the peptone stage. In 1909 Neubauer and Fischer⁴ announced that the cancer ferment had the power of hydrolyzing simple peptids. One of the products of this cleavage when the dipeptid glycyl-tryptophan is acted upon is tryptophan, an amino-acid whose presence in gastric contents is readily detected. The "glycyl-tryptophan test" of Neubauer and Fischer depends upon this reaction.

Weinstein⁵ maintains that the addition of glycyl-tryptophan to the stomach contents is unnecessary because the cancer ferment will convert the peptones of the test meal itself into amino-acids, among which is tryptophan. Accordingly he uses a modification of the glycyl-tryptophan test in which the filtrate from the test meal is tested directly

¹ Krauss, v. Graff, and Ranzi. *Wien. klin. Woch.*, 1911, No. 28.

² Weil. *Jour. Amer. Med. Assoc.*, 1908, p. 158.

³ Elsberg. *Jour. Amer. Med. Assoc.*, March 27, 1909.

⁴ Neubauer and Fischer. *Deutsch. Archiv. f. klin. Med.*, 1909, xciii, 499.

⁵ Weinstein. *Jour. Amer. Med. Assoc.*, 1910, lv, 1085.

for the presence of tryptophan without the previous addition of glycyl-tryptophan. This is known as the tryptophan test.¹

These tests have been studied by Friedman,² Hall and Williamson,³ Kohlenberger,⁴ Lyle and Kober,⁵ Oppenheim,⁶ Sanford and Rosenbloom,⁷ Smithies,⁸ Warfield,⁹ Weinstein,¹⁰ and others. A considerable difference of opinion prevails among them in regard to the factors which may be considered to invalidate the tests. The presence of blood, bile, regurgitated duodenal contents, swallowed saliva, bacteria, or of low or high acidity, or anacidity, are regarded variously as rendering them worthless or having no effect whatever on the result. No more uniform are the conclusions regarding the practical value of the tests. As is so often the case when new methods are on trial, the earlier reports are the more enthusiastic.

Smithies¹¹ has presented a valuable report of a most extensive series of cases of gastric disorders in which a routine application of modifications of both tests was made in each case. In his series, of all of the proved cases of cancer of the stomach more than one-third gave positive glycyl-tryptophan, and one-thirteenth, positive tryptophan reactions, but in each of these cases the diagnosis was quite possible independent of these methods. While in many cases of gastric conditions other than cancer a positive glycyl-tryptophan reaction was obtained, still in no single class of diseases of the stomach was the test obtained so frequently as in cancer. He concludes also that in cases of cancer of the stomach the glycyl-tryptophan reaction appeared more consistently than did the tryptophan reaction. While it cannot be stated that these tests will never prove to be of any value, still the statistics thus far reported make it clear that at the present time they are unreliable and of no assistance in the early diagnosis of gastric cancer.

Salomon's Test.—Salomon's¹² test depends upon the recovery from the stomach washings of a larger amount of albumin and nitrogen than is

¹ For details of the technique of the glycyl-tryptophan and tryptophan tests see Lyle and Kober, *New York Med. Jour.*, June 4, 1910, xci, 1152, and Weinstein, *Jour. Amer. Med. Assoc.*, October 28, 1911, lvii, 1424.

² Friedman. *Archives of Diagnosis*, 1911, iv.

³ Hall and Williamson. *Lancet*, March 18, 1911, 731.

⁴ Kohlenberger. *Deutsch. Archiv f. klin. Med.*, 1910, xcix, 148.

⁵ Lyle and Kober. *New York Med. Jour.*, June 4, 1910, xci, 1152.

⁶ Oppenheim. *Deutsch. Archiv. f. klin. Med.*, 1910-11, ci, 293.

⁷ Sanford and Rosenbloom. *Archives of Int. Med.*, April, 1912, ix, 450.

⁸ Smithies. *Archives of Int. Med.*, October 15, 1912, x, 357.

⁹ Warfield. *Bull. Johns Hopkins Hosp.*, May, 1911, 150.

¹⁰ Weinstein. *Jour. Amer. Med. Assoc.*, October 28, 1911, lvii, 1424.

¹¹ Smithies. *Archives of Int. Med.*, October 15, 1912, x, 450.

¹² Salomon. *Deutsch. med. Woch.*, 1903, No. 31.

normally present. The tissue destruction in gastric cancer is supposed to be accompanied by the pouring out of an albuminous serum.¹ Since the announcement of this test in 1903 many have endeavored to establish its true value. It is conceded to be of some value in assisting in the diagnosis of extensive ulceration, but of very little, if any, in differentiating benign from malignant conditions, especially when the latter are of the diffuse infiltrating type. This applies also to the several modifications of the test that have been proposed.

Reissner has proposed a test in which the amount of chlorides in the stomach contents is estimated. He believes that in cases of cancer the amount of chlorides poured out into the stomach is much increased over the normal amount. This test has not been demonstrated to be of value.

Livierato² has proposed to inject subdurally into previously sensitized animals, thoroughly filtered gastric juice from suspected cases, being led to believe from previous experimentation that those animals injected with gastric juice from cancerous stomachs will show symptoms of anaphylaxis, while the others will not.

This and the other almost innumerable tests of various kinds that have been proposed for the early diagnosis of gastric cancer are subject to too many important sources of error to be of any value.

COMPLICATIONS

Perforation.—An important group of complications due to perforation of the growth occurs in a little over 4 per cent. of the cases. Perforation if not limited by adhesions about the base of the growth may excite a general peritonitis, but if the leakage is slight or limited by pre-existing adhesions, the chronic form of perforation occurs, leading to the formation of localized abscesses.

General Peritonitis.—If acute perforation occurs late in the course of the disease when the patient is seriously prostrated, it may be attended by symptoms that are far less conspicuous than those which ordinarily attend perforative peritonitis. The actual perforation may be preceded by an increased pain and distress more or less constant in character

¹ A preliminary non-albuminous fluid diet is given for twenty-four hours. The evening before the stomach is carefully washed. Next morning the stomach is again washed thoroughly with 400 c.c. of normal saline solution, the same fluid being used repeatedly. The fluid used is then tested for albumin and nitrogen by Esbach's and Kjeldahl's methods. More than 0.5 gm. of albumin or 20 mg. of nitrogen to 100 c.c. of the fluid is said to be suggestive of cancer. In negative cases there should be little or no turbidity with Esbach's reagent, and the nitrogen (Kjeldahl) is not greater than 15 mg.

² Livierato. Die Magensaft; Anaphylaxie; Anwendung dieselben zur Diagnose des Magenkrebses.

and associated with tenderness and possibly with rigidity of the upper abdominal wall. In other instances hematemesis from the sloughing of the mass may precede the accident. It is usual at the time of the perforation for the patient to experience sharp agonizing pain in the epigastrium which rapidly becomes diffused over the entire abdomen, but in cases in which the peritoneum has been previously implicated by malignancy, as well as in those patients who are greatly exhausted by the disease, the pain may be slight or even absent, or may not differ in the least from pains previously experienced. In these advanced patients, however, there is a marked change in their appearance. They look sick; the nose is pinched, the eyes receded into their sockets, the surface of the body is cold, slightly cyanotic, and may be covered by beads of cold perspiration. The facies is often typical of acute septic peritonitis. In almost all cases there is general distention of the abdomen, with tenderness and rigidity, although these signs are not invariably present. It is rare for the patient to live more than three days after the accident.

Localized Perigastric Abscess.—Localized perigastric abscess occurs more frequently than does complete and sudden perforative peritonitis. The pathological anatomy of such an accident is practically that of incomplete localized perforation of ulcer, and need not therefore be given in detail in this connection.

Abscesses of the anterior abdominal wall communicating with the stomach are of infrequent occurrence, only about 25 instances of cutaneous gastric fistulæ being recorded.

Subphrenic abscess is not uncommon. The lesser peritoneal sac may be full of foul-smelling pus, and may contain air. Secondary infections above the diaphragm may occur, fibrous-serous pleurisy, empyema, or even abscess or gangrene of the lung, even though there be no metastases in the thoracic viscera. Pneumothorax or pneumopericardium may follow perforation of the esophagus by cancerous ulceration that has invaded this structure from the cardia.

Symptoms.—The symptoms of localized perigastric abscess depend upon the stage of the original gastric complaint in which the complication occurs and upon the intensity of the local inflammatory process. An increase in the pain is usually noted, not only in intensity, but in its constancy. Constant epigastric or abdominal pain in cancer, especially if accompanied by leukocytosis or a relative polynucleosis usually is suspicious evidence of abscess or sinus formation. Circumscribed abscesses that are in contact with the anterior abdominal wall usually give rise to a visible or a tangible tumor which shows fixation and undergoes no respiratory excursions. The abdominal wall over such an area is usually exquisitely tender and quite rigid.

Subphrenic abscesses may give rise to considerable dyspnea, nausea, vomiting, with repeated retching and fairly constant pain. Physical examination usually shows considerable bulging of the hypochondria, and a limitation of the respiratory movements of the lower thorax of the affected side. A characteristic tympany or even an amphoric note may be elicited by percussion; metallic tinkle may be distinctly audible. The apex of the heart in left-sided subdiaphragmatic abscesses may be displaced upward and to the left.

Fever is usually persistent in all forms of extragastric suppuration and assumes a septic type. Leukocytosis or polynucleosis is frequent. In a case of the writer's of subphrenic pyropneumothorax the leukocytes were 7000, but on differential count the polynuclears were 93 per cent.

In suspected cases the vomited matters should be examined for pus cells, as in almost all instances of incomplete perforation they are quite abundant and afford material aid in diagnosing the presence of this complication.

Although in the great majority of instances perigastric abscesses may be suspected or even correctly diagnosed, it not infrequently happens that the discovery of the abscess at autopsy is quite unexpected, and on looking over the clinical notes of the case all the evidence it gave of its presence was that the patient looked more ill, and weaker and more prostrated, ran a little temperature, and passed rapidly into a downward course toward dissolution.

Fistulas May Form.—Cutaneous fistula is quite infrequent. Gastrocolic fistulas are more common and are said to occur in about 2 per cent. of cases of cancer of the stomach, although the writer thinks that this estimate is altogether too high.

Symptoms.—The two important symptoms are:

1. Fecal vomiting.
2. Lienteric diarrhea.

Either or both of these symptoms may be absent.

In 45 cases collected by Edsall (*Amer. Med.*, October 10, 1903), fecal vomiting was present in 29 and absent in 16 cases. The vomiting may be preceded by pain, or a feeling as if something had given way and usually comes on suddenly and not gradually, as in peritonitis or obstruction. The vomited matters are evidently from the large intestine, brown and distinctly fecal, and exhibit the same characteristics as the bowel evacuations.

It is important to distinguish between the true fecal odor and a foul odor due to ulceration and sloughing of the cancer mass. Feculent vomiting is not, however, characteristic of gastrocolic fistula even in the absence of intestinal occlusion or paralysis, as it may occur in

hysteria, and even in the course of cancer of the stomach, fecal vomiting may occur without either intestinal obstruction or gastrocolic fistula. It is supposed that the cause for the fecal vomiting in these latter cases is a rigid patency of the pylorus, from cancerous infiltration, so that it is unable to contract sufficiently to prevent the passage through it of intestinal contents forced backward by a process of reversed peristalsis.

It not infrequently happens that should a gastrocolic fistula form during the course of pyloric cancer, the vomiting characteristic of pyloric stenosis may cease and an apparent improvement in the patient's condition may be quite noticeable. "The patient vomits into his own intestine," but the improvement is only temporary. Continued nausea usually accompanies such a condition.

Fecal vomiting may be absent if the fistulous opening is too small to allow the fecal masses to get into the stomach. It may also happen that fecal contents may enter the stomach, and instead of being vomited reënter the bowel through the pylorus or through a gastrojejunostomy opening. In a case reported by Kelling the distended stomach squeezed the colon walls together to form a valve so that the entrance of the stomach contents through the colon was prevented. Severe diarrhea with evacuations containing large quantities of totally undigested food passed soon after its ingestion, is observed if the false passage be of sufficient size. Rapid emaciation naturally ensues. These characteristics of the stools are frequently overlooked.

Diagnosis.—The diagnosis may be corroborated by the finding of colored fluids introduced by rectum, in the stomach or in the vomited matters, and conversely by the rapid passage of colored fluids taken by mouth through the colon and appearing in the stools, often within the hour. Inflation of the bowel by air does not distend the ascending portion of the colon, but inflates the stomach, and the air is eructated with a distinctly foul and fecal odor.

X-rays should show the lesion clearly and should definitely establish the diagnosis without any loopholes for doubt.

Metastases.—The symptoms of the various metastases are too well known to require more than the briefest reference.

Metastases in the liver usually overshadow in growth the primary source of disease in the stomach and give symptoms predominating those of gastric origin. In many instances the original cancer in the stomach becomes quiescent and clinically latent, as soon as metastases form in the liver.

The advent of *cancerous peritonitis* is usually insidious. In half of the writer's cases of this complication there was neither pain, rigidity, nor any fever, and in the majority of these the swelling of the abdomen

was noticed by the patient before there was complaint of any discomfort whatever. In but one-third of the cases was pain a prominent feature. When pain is present it may be of a dull character distributed over the entire abdomen, or the pain may be sharp, lancinating, and even colicky. Occasionally radiation down the leg may be observed.

General Complications.—*Pneumonia* is not an uncommon complication and usually constitutes the terminal event. *Aspiration pneumonia*, *gangrene* or *abscess of the lung* may occur, being especially frequent with cancer of the cardia that has extended into the esophagus. *Polyneuritis* may give rise to severe peripheral pains, usually accompanied by an increase in the reflexes, although the reflexes may be diminished or even absent. *Thrombosis*, usually of the larger veins in the leg or thigh, is not uncommon in cachectic patients, and may even occur before the appearance of gastric symptoms.

There are cases not uncommonly observed which assume the type of a simple ascites, common to affections of the heart, liver or kidneys. The patient will complain of loss of strength and occasional vomiting, but without any symptoms that point toward any serious disease of the stomach itself. The abdomen is distended with fluid, but there is neither characteristic pain on pressure nor any other abdominal sign of cancer. In many of these cases signs of pleuritic fluid are present. Such a history is as follows:

F. H., aged fifty-six years. Patient has been a steady drinker of beer—about two quarts a day, and for the past year has taken whisky with his meals. His previous history was good until three weeks ago, when he noticed his abdomen was getting large. About two weeks ago the swelling of the abdomen was much more noticeable, but the only complaint was a slight feeling of distention as if his clothes were too tight for him. One week ago he became a little short of breath and lost his appetite. At no time has he had pain, nausea, nor any vomiting.

Physical Examination.—A poorly developed emaciated man with a symmetrically enlarged abdomen which showed by examination the presence of free fluid. There were signs of fluid at both bases behind, heart negative, urine normal. No masses could be felt in the abdomen, nor any areas of tenderness. His red blood cells were 4,500,000, hemoglobin 70 per cent., white blood cells 6000, 66 per cent. polynuclears. The course of the disease after admission to the hospital was steadily downward, the chief symptoms being swelling of the abdomen, edema of the ankles, slight dyspnea, and occasional vomiting of his medication. Death occurred one month after he entered the hospital.

Autopsy showed free fluid in the abdominal cavity. The stomach was adherent to the liver, diaphragm, intestines, pancreas, and spleen.

Its walls were diffusely thickened, and its capacity markedly diminished, the so-called "leather-bottle" stomach. The mesentery, omentum, and abdominal peritoneum were studded with small carcinomatous growths, and the intestines were matted together by adhesions. There was fluid in both pleural cavities and a small area of pneumonia in the upper lobe of the right side. There were small metastases in the heart muscle.

Phlegmonous gastritis may involve the wall of the stomach in the neighborhood of an ulcerating mass. The phlegmon usually remains circumscribed, and but rarely gives symptoms of its own, as the complication is apt to occur only in the advanced stages of cancer when the patient is too weak and prostrated for this complication to make any decided difference in the clinical course of the disease.

DURATION, DIAGNOSIS, AND TREATMENT

Duration.—The duration of the disease is about one and a half to two years, being somewhat shorter in the young than in the aged. The actual duration is, however, difficult to determine, because in some patients the symptoms begin early and in others they do not appear until the disease is well advanced. So that it is impossible to estimate how long the cancer has existed in any given case before it has reached the point of giving symptoms. It may happen that patients run a clinical course of only a few weeks, while others suffer for many months from the malady.

Diagnosis.—The important problems of diagnosis are concerned with the detection of the growth in its early stages while there is a chance that the disease can be radically removed by timely operative intervention. It makes very little difference in advanced inoperable cases whether we make a diagnosis or not. The difficulty which confronts us in the early cases is that if we wait for characteristic or typical symptoms to clear up the diagnosis in a suspected case, we have probably waited too long for surgical relief to be radical and curative, while, on the other hand, if we advise exploration before we have established our diagnosis, we are often subjecting our patients to a totally unnecessary operation.

Diagnosis of Early Cases.—The early cases may be divided into two groups—those who give an ulcer history, and those in whom the symptoms appear without any previous complaint of indigestion.

(a) *Early patients with an ulcer history.*

In many patients the first indication of trouble with the stomach is the occurrence of a group of symptoms which may so closely resemble

ulcer that a differentiation is practically impossible. The pains may occur two or three hours after eating and are relieved by eating, as are the pains of ulcer. Gastric analysis shows no essential differences in these two conditions.

The best method of making a differential diagnosis in these cases is to put the patient at once on a rigid ulcer cure. He should be put to bed with hot applications over the abdomen, and a total abstinence of food or drink should be enforced for seventy-two hours. At the end of this time peptonized milk is given in small quantities according to the treatment given in detail under the treatment of ulcer. Ordinarily on this treatment pain and discomfort cease before the tenth day, the stools become blood-free, and although there is an initial loss from the starvation treatment from 6 to 8 pounds, the patient after ten days should begin to gain a little. If in the case in point the patient complains of a continuance of his ulcer symptoms, if blood is present in the stools during the second and third week of the ulcer cure, or should blood in the stools reappear with each advance in his diet, if the patient should not regain flesh and strength in the third and fourth week when his diet is sufficient to enable him to gain, or if during the third and fourth week blood examination shows an increasing chloranemia in spite of sufficient food, then the case should be regarded as suspicious, a surgeon of good judgment and irreproachable technique should be called into the case and the question of exploratory laparotomy seriously discussed. These points have already been alluded to under the heading of ulcer.

In other patients there may be elicited the history of an ulcer in the past, the symptoms of which have become quiescent so that it may be considered that the patient is cured of the original disorder. Should such a patient complain of an unexplained or sudden loss of appetite, or loss of weight, together with vague dyspeptic symptoms which cannot be explained by dietetic errors, then the diagnosis of a carcinomatous degeneration of an old unhealed ulcer must be seriously considered. Under these circumstances it is wise to place the patient on a gastritis treatment, to give him a light suitable diet, in quantity and quality adapted to his special needs, and if necessary to wash the stomach every day. Frequent blood counts should be taken and the record of his weight duly recorded. If after a month of such treatment, faithfully and conscientiously carried out, there be no material gain, it is generally wise to explore, to determine the exact conditions that are preventing restoration to health.

A large proportion of these patients are treated by the physician for chronic gastritis. Especially is this diagnosis made when vomiting occurs or if the patient complain of discomfort in the region of the

stomach. It cannot be too strongly emphasized that vomiting and pain in the stomach are not symptoms of chronic gastritis in those of adult years, and that a diagnosis of gastric catarrh should never be made on the presence of these symptoms. Nor should cancer of the stomach be discarded purely on the basis of age. Many cases are not recognized simply because they occur in young people. We are often misled in our diagnosis by the persistence of good appetite and a lack of anemic or cachectic symptoms. It not infrequently happens that the patient retains a robust appearance and has a normal appetite until the disease is well established.

During the early stages of cancer, gastric analysis may or may not be of service. The most significant sign of disease is the presence of fluid or of food remains in the fasting stomach, indicative of motor error. Such stasis may occur from benign stenosis as well as from malignant disease of the pylorus, but in a case where the symptoms are obscure and ill-defined, an increasing food-stasis is highly suspicious of an advancing malignancy. The absence of hydrochloric acid means nothing. Its presence is common enough in early growths and in instances in which carcinomatous degeneration is implanted on the base of an old ulcer it may persist throughout the entire course of the disease. It is unfortunate that in the minds of many an absence of hydrochloric acid in the test breakfast is regarded as a suspicious sign of malignancy. Of 153 consecutive cases of absence of hydrochloric acid in the test breakfast in the writer's private cases, 4 were malignant, 149 were non-malignant. It cannot be too positively affirmed, therefore, that hydrochloric acid values have very little bearing on the diagnosis of gastric cancer.

On the other hand, achylia with stagnation justifies exploration.

The presence of lactic acid is an extremely suspicious phenomenon though not pathognomonic of cancer of the stomach itself. It usually is a late symptom, and, as a rule, indicates that the disease is too firmly established for any hope of radical relief. It is improbable that gastroscopy will ever be of any aid in the diagnosis. The various chemical, biological, and hemolytic tests suggested for the diagnosis of malignant neoplasms may corroborate the diagnosis of advanced stages of the malady, but are not to be relied upon for diagnosis in the early cases.

Late Cases.—In advanced stages of the disease there is usually very little difficulty in making a diagnosis, as we have the history of the case, the physical examination and the gastric analyses to aid us. No one of these alone is sufficient to justify a diagnosis. Even the demonstration of a tumor does not prove that it is malignant, but the combination of any two of these three forms of examination usually renders the diagnosis quite evident. It is not enough, however, to be satisfied

with the statement that the patient has a cancer of the stomach. The location of the growth must be determined, in reference to either radical or palliative operation, and the extent to which the disease has invaded other parts must be determined before submitting the patient to the pain and anxieties of a futile operation.

Treatment.—The treatment of cancer of the stomach should be entirely surgical. There is no doubt that this would be actually the case if we were sufficiently skilful to make the diagnosis of cancer in time. Unfortunately either from our own inability to interpret symptoms correctly or because the patients come to us in a too advanced stage of the malady, it usually happens that the diagnosis is too late for any other treatment than that purely palliative and symptomatic. In these late cases diet, lavage and other medical means are often of service in reducing the severity of the symptoms and improving the general condition of the patient, and in these respects they are useful as far as they go.

Diet.—There is no standard diet for cancer of the stomach. Each patient must be advised according to the indications of his own particular case. The quantity of food at any one meal is to be determined by the degree of food stasis that may be demonstrated in the case, small frequent feedings being advisable in those patients whose stomachs show evidence of motor error. The quality of the food on general principles should be bland, unirritating, and wholesome. Due regard should be paid to the caloric values to insure a sufficiency for body gain. Due respect will have to be paid to the caprices of the appetite, and food that is theoretically contra-indicated but craved by the patient may agree far better than unappetizing articles of diet prescribed in accordance with a definite routine.

As a rule meats are poorly digested, and for this reason beef and the heavier forms of meat should be allowed sparingly. Fish and chicken may be given in their stead.

In the advanced cases that come under treatment with the history of repeated vomiting it is usually best to begin with a purely milk diet, to which may later be added malted milk, custard, junket, and the finer cereals. Later the diet advised for the third and fourth week of ulcer cure may be given (see page 183).

Drugs.—Three classes of drugs may be employed.

1. *Those Directed Toward the Correction of Errors of Secretion.*—If the fasting stomach contains free hydrochloric acid fluid in excess, or if the test breakfast show an alimentary hypersecretion, alkalies are indicated to neutralize the excess of acid. Any form of alkalies commonly in use may be used—bicarbonate of soda, magnesia usta, or the use of alkaline waters, such as Celestins Vichy.

When gastric analysis shows a diminution or absence in hydrochloric acid, artificial means for digestion may be employed. Hydrochloric acid is often of service given with or directly after the meals. It may be judiciously combined with bitter tonics, such as gentian or the fluid extract of condurango. Oxyntin with pepsin (Fairchild) may be given in 10-grain doses in capsule with the meals, or tablets of acidol in the same doses. Pepsin as ordinarily prescribed is often inert. Those preparations of the ferment which are physiologically active have failed in the writer's experience to be of any material service. More serviceable than pepsin is pancreatin or pancreon, preferably given admixed with alkaline powder in cases in which hydrochloric acid is absent from the test breakfast.

2. *Drugs to Improve the General Nutrition.*—There is very little use in giving iron or tonics or in giving bitters, such as condurango, to improve the appetite, and yet these drugs are prescribed not because we expect any good from their use, but because they serve to buoy up the patient and to allay in part the anxiety of the family.

3. *Drugs to Relieve Pain.*—Pain may be relieved in the hyperacid cases by the use of alkalies, as in the case of ulcer. The pain that is due to increased peristalsis from pyloric closure is best treated by lavage, by regulation of the diet, and by the use of olive oil before meals. A very useful preparation is a 3 per cent. solution of anesthesin in olive oil, the dose of which is one to two tablespoonsful before eating. Orthoform does not relieve the pain of cancer as it does the pain of ulcer. Atropine as a controller of pain has not been of service in the writer's cases. Sooner or later we are obliged to resort to opium, or some of its derivatives. Codeia presents the least disadvantages and should be first employed. When codeia fails the writer has relied upon the following prescription:

R—Pulv. opii denarcot.	gr. ss
Pulv. aromatic.	gr. ivss
M. ft. caps. no. j.	
Sig.—One two or more times a day for pain.	

It has seemed as though opium in this form was more easily tolerated than was morphine. In very severe cases reliance will have to be placed upon morphine given hypodermically.

Lavage.—Lavage is indicated when food-stasis exists and should be repeated daily either before breakfast or late in the afternoon before the last meal of the day. The greatest gain derived from lavage is the improvement in subjective symptoms. The patients feel better, eat better, and suffer less discomfort from their meals. When no actual stasis can be demonstrated, lavage may be of some benefit without

our knowing why it should do good. In the majority of cases lavage with plain water is to be employed. The addition of antiseptics is rarely of service, although when the mass is sloughing or the gastric contents are foul, such indication may diminish the fetor and improve the appetite. Resorcin, gr. x, or essence of peppermint in sufficient quantities to make the lavage water pleasantly aromatic, has in the writer's experience been among the most efficient.

Surgical Treatment.—It is after all not an objection to surgical treatment that the operative mortality is as high as it is. Instead of considering that 25 or 30 per cent. of patients die from their operation we should congratulate ourselves that the balance survive the operation and are either benefited temporarily or stand chance of a more or less permanent cure. The mortality of surgical treatment is difficult to estimate because knowing that the disease is inevitably fatal without an operation some operators take greater chances than others, some operate more radically than others, and while the more radical and bolder operators have a higher percentage of immediate mortality, nevertheless they often succeed in postponing the fatal event. Accordingly the mortality varies from 14 to 40 per cent., with a general average of 25 per cent. as an immediate result of operation for the radical cure of gastric carcinoma.

The surgical treatment may be either exploratory, radical, or palliative.

Exploratory incision is accompanied by a minimum of risk, and should be resorted to in every doubtful case in which there is any suspicion whatever of malignancy. It is not wise to tell the patient the reason for the operation, although some member of the family should be taken into confidence.

Radical operation is to be done whenever feasible, and if any error is made it should be by taking away too much rather than too little of the affected area.

Palliative operations are indicated for the relief of pyloric stenosis. Gastrojejunostomy in these instances is frequently followed by a most brilliant improvement for the time being. Not only are the symptoms of food-stasis relieved, but the patients gain in weight and appetite, and even in extensive growths the pain may become almost negligible. It not infrequently happens that after such a gastrojejunostomy for malignant stenosis the patient will eat everything with impunity, will gain 30 to 40 pounds in weight, and will be able to do his daily work. After a certain period of time, but often after months, the symptoms will return and the patient will suddenly lose flesh and strength and die from extreme weakness.

CHAPTER VII

SARCOMA OF THE STOMACH

SARCOMA of the stomach is comparatively infrequent, probably not exceeding 1 per cent. of gastric tumors. Tilger in 3500 autopsies found but a single case, while Hosch in 13,387 autopsies encountered 6 instances. To show how infrequent is the involvement of the stomach in sarcoma, of 1263 autopsies on sarcomatous patients, compiled from pathological records in Munich and Berlin, by Wilde and Gurlt, there was not a single instance in which the stomach was involved.¹ Fenwick, however, considers that sarcoma is somewhat more frequently observed than is supposed, and estimates that 5 to 8 per cent. of gastric tumors are of this nature, basing this rather high estimate upon the fact that in the earlier days of accurate pathology neoplasms were regarded as cancer that should have been correctly diagnosticated as sarcoma. His estimate is corroborated by Perry and Shaw who found 4 cases of sarcoma in 50 instances of malignant disease of the stomach.

The writer believes that this estimate of Fenwick's is entirely too high, for in 1910 only 123 cases were reported, and Gossett,² has been able to collect but 171 cases of gastric sarcoma.

Both sexes appear to be equally susceptible to the disease.

Sarcoma shows a more even distribution among the ages than cancer, and among young subjects lymphosarcoma and round-cell sarcoma are especially frequent. It is wrong, however, to consider that this form of neoplasm is confined to the young, as it is actually more common in adult years. The age of the patients recorded by Ziesché and Burgaud is as follows:

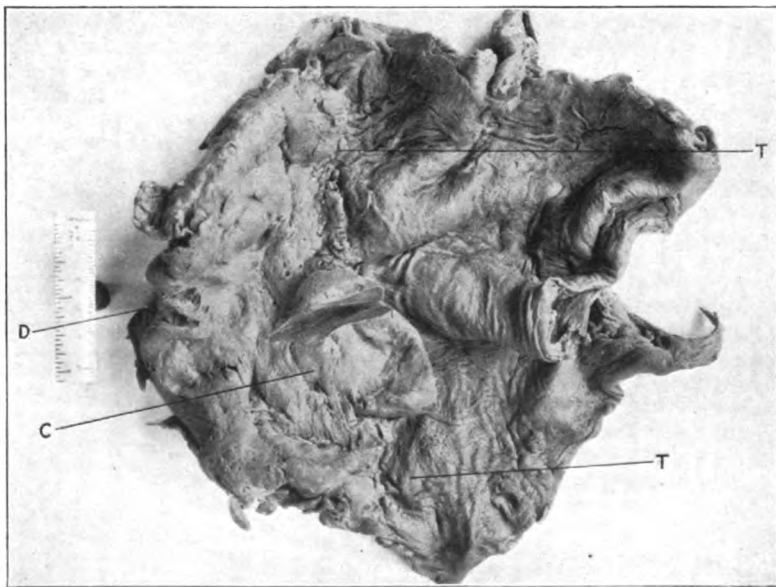
Age.	Ziesché. Cases.	Burgaud. Cases.
0 to 10 years	2	2
10 to 20 years	11	7
20 to 30 years	18	10
30 to 40 years	15	14
40 to 50 years	29	19
50 to 60 years	24	18
60 to 70 years	12	10
70 to 80 years	6	5
	<hr/> 117	<hr/> 85

¹ These writers presented separate reports from their respective laboratories. The two sets of statistics are here combined.

² La Presse Médicale, March 16, 1912.

Pathology.—Gastric sarcoma may occur either as a circumscribed or a diffuse infiltration, which almost invariably is the case with lymphosarcoma and the round-cell sarcoma, or it may appear as a tumor, either in the wall of the stomach, or projecting as a pear-shaped, pedunculated growth into the lumen of the stomach, or outwardly into the greater or lesser peritoneal cavity, but connected with the stomach wall by a pedicle. The tendency to grow inwardly or outwardly has led to the subdivision of such pedunculated growths into the endogastric and the exogastric form, and these growths are largely made up of the spindle-cell form. The newgrowth usually arises in the submucosa or the muscularis, the mucous membrane being involved late in the disease, if at all. In many cases the mucous membrane slides normally over the tumor mass, though in some instances it may be adherent and sloughing.

FIG. 45

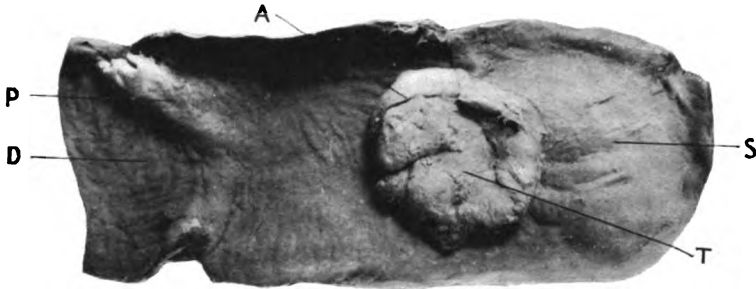


Sarcoma of the pyloric end of stomach. The tumor is fairly sharply outlined at *T, T*; main mass of tumor is seen at *C*; *D*, duodenum. (From the Pathological Museum, Mt. Sinai Hospital, New York.)

Sarcoma of the stomach may be primary or secondary. Primary sarcoma is the ordinary form, and is nine times as frequent as the secondary form. Secondary sarcoma is usually melanosarcoma or lymphosarcoma. The spindle form is rare. Secondary deposits may be metastatic, arising from a primary growth in the neck, pharynx,

rectum, or in the retroperitoneal tissues, or in other parts of the body, or they may occur as part of the process of general lymphosarcomatosis of the gastro-intestinal tract. In other instances the secondary nodules represent the internal manifestations of Hodgkin's disease.

FIG. 46



Lymphosarcoma of the stomach. *D*, duodenum; *S*, stomach; *P*, pyloric ring; *T*, tumor. At *A* it is seen emerging from the mucous membrane. (From the Pathological Museum, Mt. Sinai Hospital, New York.)

All varieties of sarcoma may occur in the stomach. In 117 cases collected by Ziesché and Davidsohn the following varieties were encountered.

Round-cell sarcoma	35 cases
Spindle-cell sarcoma	26 cases
Lymphosarcoma	23 cases
Myosarcoma	20 cases
Mixed sarcoma	2 cases
Angiosarcoma	5 cases
Lymphangio	3 cases
Myxosarcoma	3 cases

The frequency in which these varieties occur is given somewhat differently by Fenwick, who has found round-cell sarcoma in 62 per cent. of the cases, and considers that myosarcoma is extremely rare.

The situation of the growth is more widespread than is the case with cancer, as is seen in the analysis of the implantation of the growth in 100 cases reported by Ziesché and Davidsohn:

Pylorus	25 cases
Greater curvature	22 cases
Diffuse infiltration	18 cases
Posterior wall	15 cases
Lesser curvature	11 cases
Anterior wall	6 cases
Fundus	1 case
Cardia	2 cases

Tumors arising from the greater curvature are often pediculated and extend outwardly from the stomach, assuming the exogastric form.

Although 25 per cent. of sarcomas involve the pylorus, actual obstruction of that orifice is somewhat unusual.

The size of the growth varies from nodules barely visible to the naked eye, up to large tumor masses the size of a child's head, or even larger, and often weighing over twelve pounds.

Various degenerations of the growth may occur. Of these hemorrhage into the tumor substance and cystic degeneration are the most frequent. Some of the cysts of the larger sarcomas may contain several liters of fluid.

Metastases are not only less frequent than in cancer, but are less liable to be multiple. There is, moreover, not the same tendency to invade contiguous parts by direct extension, as is the case with cancer. Metastases are more common in the round-cell sarcoma and lympho-sarcoma than with the spindle-cell form.

According to Fenwick, metastases are found in 70 per cent. of the round-cell variety:

Lymph glands		50 per cent.
Kidneys		28 per cent.
Liver		
Ovaries	} each	
Pancreas		
Adrenals		
Omentum		14 per cent.
Skin nodules		12 per cent.
Lungs		
Diaphragm	} each	
Pleural		
Esophagus		
Intestines		7 per cent.
Mesentery		

Again we have a difference between Fenwick's and Ziesché's figures, for of 84 cases of metastases reported by these latter writers, including, however, all varieties of sarcomatous growths, there were involved:

Lymph glands	23 cases
Liver	18 cases
Intestines	7 cases
Ovary	5 cases
Mesentery	5 cases
Pancreas	4 cases
Skin nodules	4 cases
Kidney	3 cases
Diaphragm	3 cases
Bones	3 cases
Spleen	2 cases
Scattered	7 cases

It is interesting to note the large proportion of cases showing metastatic deposits in the skin. These skin metastases vary in size from a head of a pin to a bean, and they may be excised and examined, establishing the diagnosis.

In a few instances the neoplasm has implanted itself on the site of an unhealed ulcer, and occasionally sarcoma of the stomach has followed local injury, as in the case of Brooks, where sarcoma developed in the cicatrix of a bullet wound of the lesser curvature. Sarcomatous degeneration of myoma or fibromyoma of the stomach may occur and these mixed forms often attain considerable proportions.

Symptoms.—The symptoms are general and local.

General Symptoms.—Cachexia is usually well-marked early in the disease, and constitutes a most striking feature of the complaint. Progressive loss of flesh with failure of physical powers is usually conspicuous even in the early cases, and is especially noticeable when a round-cell growth has involved the pylorus. Anemia is always present, and gradually increases, so that the pallor may be as marked as that met with in pernicious anemia. The blood shows the characteristics of the chloranemia or even the chlorotic type of anemia. The hemoglobin may be reduced to 15 or even 10 per cent. of the normal. The number of the red cells is reduced but not in the same ratio. In one of Mange's cases there were 4,000,000 reds and 30 per cent. hemoglobin. Leukocytosis occurs only with complications or with the ulceration of the mass.

Pyrexia may be observed in young subjects or in those in which the neoplasm grows rapidly or undergoes degeneration. The febrile reaction is apt to be low—99° to 101°—but continuous. Sharper attacks of fever accompany the development of complications. Albuminuria is present in about one-sixth of the cases, especially in the round-cell variety, and usually indicates metastases in the kidney.

While these general symptoms are commonly enough observed in the majority of sarcomas, there occur more benign forms in which the general symptoms caused by the neoplasm are slight or negligible, even though the growth has attained considerable size. This is especially the case with exogastric myosarcoma. It not infrequently happens that a mass is discovered by physical examination which has given neither general nor gastric symptoms of sufficient importance to arouse suspicion of the disease. Cantwell reports finding such a growth weighing 12 pounds which had given absolutely no symptoms.

Local Symptoms.—There is practically no difference between the local symptoms of sarcoma and cancer. In the earlier stages indigestion is complained of in a vague and indefinite way, and affords no clue to the diagnosis. Especially is this apt to be the case with the exogastric

myosarcoma. Many of these exogastric growths give only the physical signs of an abdominal tumor of unknown nature and of unknown attachment, and may give no symptoms until the occurrence of such complications as adhesions, torsions, hemorrhages into the tumor, suppuration of the tumor, or metastases. Vomiting is more rare than with cancer, and is a late symptom unless the pylorus be involved by a diffuse growth.

Although the pylorus is frequently involved, the vomiting is rarely characteristic of pyloric stenosis. Hemorrhages are somewhat rarer than with cancer, although copious and sudden hematemesis may be the first indication of disease.

Hematemesis seems to be less common in the round-cell variety, which rarely causes ulceration of the mucosa, than in the spindle-cell form, in which hematemesis is quite frequently observed. It is somewhat difficult to explain the frequency of hematemesis in these exogastric tumors, as the mucous membrane corresponding to the attachment of the pedicle is usually intact.

Pain is a prominent symptom, being absent in but 4 per cent. of 150 cases reported by Ziesché and Davidsohn, although it was not complained of in 25 per cent. of Fenwick's series. There may be a sense of fulness and oppression after eating, or actual pain either dull and aching in character or sharp and cramp-like. Sudden colicky paroxysmal pains may occur closely resembling those of renal or biliary calculus. Severe and continuous pain with exacerbations of greater severity after eating usually indicates ulceration of the neoplasm, or its extension into the pancreas or retroperitoneal tissues. This sudden and continuous pain was noted in 15 per cent. of Fenwick's collected cases. Solid exogastric sarcomas, chiefly of the fibro- or fibromyomatous forms, rarely give actual pain until the growth is well advanced, but are rather accompanied by a dragging sense of weight, aggravated by exercise and relieved by recumbency.

The appetite fails as early as with cancer, although the desire for food may be retained throughout the disease.

Symptoms of volvulus may appear, occasioned by the torsion of the stomach on its longitudinal axis from the mechanical weight of an exogastric growth, but this complication is less frequent with sarcoma than with benign tumors, such as fibromas or myoma, as in sarcoma adhesions are more readily formed which serve to hold the tumor mass in place and to prevent freedom of movement downward.

Gastric Analysis.—Gastric analysis is practically that of cancer, so that a differential diagnosis by this method of examination is quite impossible.

Few examinations of the *fasting stomach* seem to have been made

in the recorded cases. Extreme degrees of food-stasis are more rarely observed than in cancer.

Test breakfast shows the same variations as in cancer. Of 18 cases reported by Ziesché and Davidsohn in which gastric analyses were made, normal chemical reactions were present in 7. Hydrochloric acid was absent, lactic present in 6, both acids present in 6, both acids absent in 3. Oppler-Boas bacilli are frequently found in the gastric contents.

Physical Signs.—A palpable tumor was found in 66 out of 72 cases in Ziesché's and Davidsohn's series. These observers group all varieties of sarcoma together and compile their statistics from the admixture.

Fenwick separates round-cell from the fibrosarcoma and myosarcoma, owing to the difference in the clinical course and physical signs of the two groups. Round-cell sarcoma produces in the majority of instances a local infiltration of the gastric wall in the region of the pylorus, which may give rise only to a localized sense of resistance and localized tenderness, or to a round or oval mass, smooth, tender, and usually quite freely movable. Fenwick found that a palpable tumor of the stomach was an inconstant sign of round-celled sarcoma and was observed in but 30 per cent. of the recorded cases. On the other hand, fibro- and myosarcoma almost invariably give rise to tumors, often so large as to fill the entire abdominal cavity.

If the growth arises near the greater curvature it may be detected in the central or even in the lower portion of the abdomen, a firm, smooth, and painless mass, quite freely movable in all directions. The extreme mobility of exogastric sarcomas is in striking contrast to cancer which more rapidly forms fixed adhesions to adjacent viscera.

The physical signs of pyloric obstruction may be frequently elicited, dilatation of the stomach, succussion sounds audible at a time when the viscus should be empty, and occasionally gastric stiffening, visible peristaltic waves, and other phenomena of hypertonus. Extreme degrees of pyloric stenosis are, however, rarely encountered, for although submucous infiltration of the pyloric canal is not uncommon in the round-cell sarcoma, it does not seem to interfere with the patency of the orifice as much as one would expect.

Enlargement of the spleen so that the edge is distinctly palpable just beyond the line of the costal arch occurs in about 12 per cent. of all cases, being due to hyperemia and hypertrophy of the viscus rather than to metastases in its substance. Splenic enlargement may be of slight service in differentiating sarcoma of the stomach from cancer. It should be remembered that in a few instances a diffuse round-cell infiltration of the greater part of the entire stomach wall has been palpable as a tumor mass projecting from under the free border of the

ribs into the hypochondrium, and has been mistaken for splenic enlargement.

Kundrat has called attention to the aid afforded in diagnosis by the presence of enlarged tonsils and occasional swelling and ulceration of the follicles of the tongue. These phenomena have been, however, noted in very few of the recorded cases.

Metastatic deposits in the skin constitute an important feature of the disease in about one-eighth of the cases. The nodules may appear about the umbilicus or scattered over the abdomen, chest, and back. At first they are freely movable, but after a time they tend to become adherent to the skin and may even ulcerate. They should always be searched for in doubtful cases, excised and examined.

Enlargement of the supraclavicular glands is rare compared with cancer. In one instance the diagnosis was made by the discovery of a secondary nodule in the rectum. Ascites may appear and obscure the abdominal signs of disease, but this complication is relatively infrequent compared with cancer, as it occurred in less than 3 per cent. of the reported cases.

Duration.—Duration depends largely upon the particular variety of sarcoma in question. Generally speaking the duration is longer than in cancer, as the tumor is less malignant in character, shows less tendency to direct invasion of neighboring parts, a diminished tendency to form metastases, and even with pyloric implantation, rarely gives rise to extreme stenosis with its attendant exhaustion and emaciation.

The average duration of life with small-cell sarcoma is about one and one-half years, with spindle-cell growths over two years, while in the exogastric forms of fibro- and myosarcoma life may be prolonged three to five years.

Death usually results from anemia and exhaustion, and is often preceded by a semicomatose state extending over several days.

Perforation of the stomach and general peritonitis occurs in about 10 per cent. of the round-cell growths, far more rarely in the other forms. Owing to the absence of extensive adhesions incomplete perforation and perigastric abscess are exceptional. Fatal hemorrhage is rare.

Diagnosis.—Exogastric growths with indefinite symptoms may be mistaken for ovarian tumors, mesenteric or pancreatic cysts, or any of the known forms of abdominal tumors. An exploratory incision is usually necessary to reveal the character of the growth and accurately locate its attachment.

When cachexia, anemia, and gastric symptoms are present it is quite evident that we are dealing with a disease of malignant nature, and the point to be decided usually is the differentiation of the disease from

cancer. Sarcoma may be suspected should the patient be under thirty-five, and the younger the patient the greater the probability that the disease is sarcomatous in character. Low continuous fever is more common with sarcoma than with carcinoma. Enlargement of the spleen is also more frequent. Large, fixed nodular tumors due to invasion of omentum and other neighboring parts, and extensive metastases of the liver are indicative of cancer rather than of sarcoma.

The differential diagnosis is almost invariably quite impossible, nor does it matter in the least whether a diagnosis between cancer and sarcoma is made before operation, as the operative indications are identical in the two conditions.

Treatment.—Treatment is that of cancer. Early exploration should be advised in suspected cases, and if possible free removal of the growth should be attempted. Extensive round-cell infiltration of the stomach wall naturally renders complete removal an impossibility, but circumscribed infiltrations and large pedunculated tumors that involve by their pedicle attachment a comparatively small area of the gastric wall, are favorable for extirpation.

CHAPTER VIII

BENIGN TUMORS AND FOREIGN BODIES

BENIGN TUMORS OF THE STOMACH

IN addition to the malignant tumors already described, one occasionally meets with benign gastric neoplasms. These tumors are rare, and are more interesting as pathological curiosities than as clinical conditions. In fact, a large proportion of the reported cases were accidental discoveries at autopsy in patients who had no gastric symptoms during life. An increasing number are being found at operation in modern surgery, and in a fair number there have been definite symptoms referable to the stomach. However, these symptoms merely indicate that the stomach is the seat of the patient's trouble. They in no way indicate that one has some rare form of benign tumor to deal with. Practically all of the various symptoms which have been reported in these cases, would most naturally be interpreted as being caused by some more common gastric trouble. Very rarely a piece of the tumor recovered from the stomach by vomiting or the tube may furnish a diagnosis.

The existence of a palpable gastric tumor which has existed long enough to rule out the possibility of malignancy may offer a clue; but here one has difficulty in ruling out a hyperplastic inflammatory mass, and it is often exceedingly difficult to be sure that a given tumor mass is connected with the stomach.

Fibromyoma.—Various cases of fibroma and myoma of the stomach have been reported, but as these tumors almost invariably show both fibrous and muscular tissue in varying proportions, it is better to consider them under one group—fibromyomas. The conditions are very much the same as with similar tumors of the uterus, in which all neoplasms showing fibrous and muscular tissue are referred to as fibromyomas. One does not ordinarily use the terms fibroid, fibroma, or myoma.

Attention was first called to these tumors in 1761 by Morgagni.¹ In 1898, Steiner² reported 21 cases. To this number Deaver and Ashhurst³ have added 28 cases.

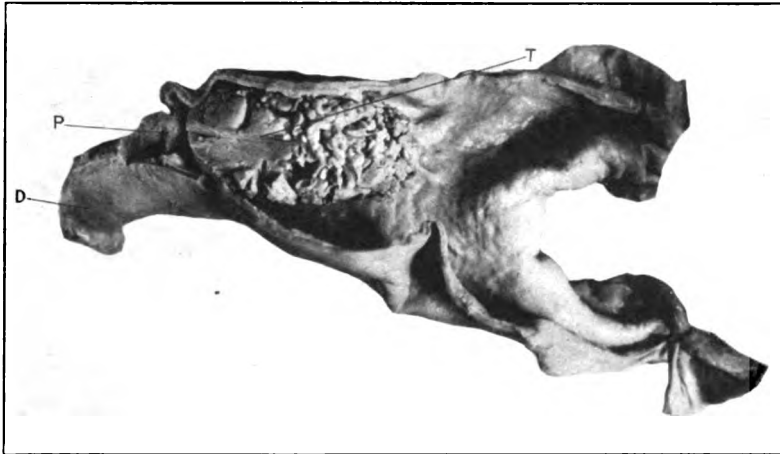
¹ De Sed. et Caus. Morb. Epist., xix, Art. 58, Venetiis, 1761, Tome i, f. 191.

² Beitr. z. klin. Chir., 1898, xxii, 1, 407.

³ Surgery of the Upper Abdomen, 1908, i, 214.

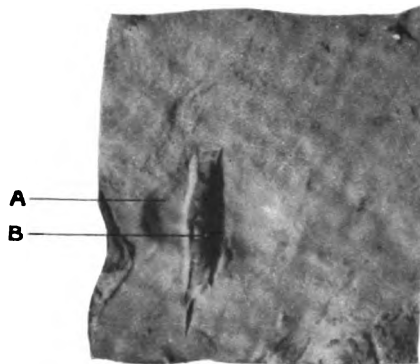
These tumors arise in the muscular coats of the stomach and, according as to whether they grow toward the interior of the stomach or externally, are divided into two fairly distinct forms.

FIG. 47



Papilloma of stomach at the pylorus. The tumor (*T*) has a broad pedicle, not shown in the picture. The tumor is so situated that it causes considerable obstruction of the pylorus (*P*). *D*, duodenum. (From the Pathological Museum, Columbia University, New York.)

FIG. 48



Fibroma of stomach. *A*, fibroma; *B*, showing portion removed for microscopical examination. (From the Pathological Museum, Columbia University, New York.)

Internal or Submucous.—These tumors are generally round or oval in shape, smooth or lobulated, brownish or dirty white in color, covered by mucous membrane and situated most commonly along one of the curvatures, generally near the pylorus. They may be pedunculated, the pedicle being often nearly as broad as the tumor. The pedunculated form may be multiple, while the ordinary form is very rarely so.

These submucous tumors are small, generally not exceeding an English walnut in size. They may become ulcerated, causing small, repeated bleeding, or even fatal hemorrhage. They may undergo cystic or myxomatous degeneration, or show malignant changes, with metastases.

Microscopically, fibromyomas show interlacing bundles and whorls of unstriped muscle and fibrous tissue, arranged in more or less concentric layers. The proportion of the two kinds of tissue varies greatly. The tumor is covered with mucous membrane, and may show superficial ulceration.

Symptoms.—The location of the tumor and the condition of its free surface determine the character of the symptoms. When located away from the stomach orifices, and when not ulcerated, these tumors rarely give any symptoms. If the tumor is situated at the cardiac orifice the patient may suffer from dysphagia, while obstruction at the pylorus gives the symptoms of pyloric stenosis and subsequent dilatation of the stomach. The pedunculated form in this location may act as a ball valve at the pylorus, or even prolapse through the opening, causing sudden, violent attacks of pain and vomiting, lasting from a few minutes to several hours.

Ulceration of the tumor leads to hemorrhage, and one has the clinical picture of gastric ulcer—pain, hematemesis, and melena.

External or Subserous.—While it is possible for these tumors to be of small size, they are generally much larger than the preceding variety. They are firm, irregularly nodular, and of a yellowish or dirty white color. The largest ones reported weighed 5400 grams¹ and 6000 grams,² the latter reaching deeply into the pelvis.

Microscopically, they do not differ essentially from the submucous form.

Symptoms.—The principal symptoms are those of gastric distress, dragging pain from the weight of the tumor, and more uncommonly, those due to some mechanical obstruction by the tumor. There have been several cases reported in which such tumors caused volvulus of the stomach.

On examination it is often possible to palpate a tumor in the abdomen. It is not always easy to determine whether it arises from the stomach.

¹ v. Erlach, *Centralbl. f. allg. Path.*, 1895, p. 240.

² Perls and Neelsen, *Allg. Path.*, Stuttgart, 1886.

Treatment.—Operative procedures should be undertaken whenever a diagnosis can be made and the tumor removed, either by excision of that part of the stomach wall, or by partial gastrectomy.

Lipoma.—These tumors are more uncommon than fibromyoma. Virchow¹ recognized their existence, and Cruveilhier² believed that the small ones were not unusual. Deaver and Ashhurst³ give a list of the few reported cases. Like fibromyoma they may exist as subserous or submucous tumors. The former are apt to be large and form pendulous tumors on the external surface of the stomach, near the greater curvature. They may cause dragging pain from the mechanical displacement of the stomach, and it may be possible to feel the tumor through the abdominal wall. The latter occur as yellow, rounded tumors projecting from the inner surface of the stomach near its central portions. They generally do not exceed an English walnut in size. They are covered by mucous membrane. They give no characteristic symptoms.

At times either variety is distinctly pedunculated, and one very rarely sees cystic degeneration. Under the microscope these tumors are seen to be composed of fatty tissue. At times there is a fairly large amount of fibrous tissue scattered through the tumor.

Treatment.—If a diagnosis can be made, operation for the removal of the tumor is indicated.

Adenoma.—These tumors are generally divided into the two following forms:

Pedunculated Form.—In this variety, the rounded, smooth, or lobulated tumor is seen attached to the stomach wall by a pedicle, generally in the pyloric region. They are generally single. Occasionally, several of the tumors are present. In color they are brown or grayish brown, and in consistence firm. Chaput⁴ has reported a solitary tumor of this kind as large as a fetal head. When multiple they rarely exceed a walnut in size. Fenwick⁵ mentions a case in which four adenomas, each the size of a pigeon's egg, were found attached to the margin of the pyloric ring, causing partial obstruction of the orifice.

On section these tumors appear firm and smooth. Occasionally small cysts are seen. Microscopically the tumor consists of proliferating gastric glands in a connective-tissue framework, rich in bloodvessels. The mucous membrane covering the tumor shows chronic interstitial inflammation. It is rarely ulcerated. There may be enough fibrous tissue to warrant the name fibroadenoma.

¹ Path. des Tumeurs, Paris, 1867, i, 369.

² Anat. Pathol., Paris, 1835–42, Tom. II, XXXe, Livr., Pl. II, Consid. Gén. P. 3.

³ Surgery of the Upper Abdomen, vol. i, p. 226.

⁴ Bull. et mém. Soc. anat., Paris, 1895, lxx, 534.

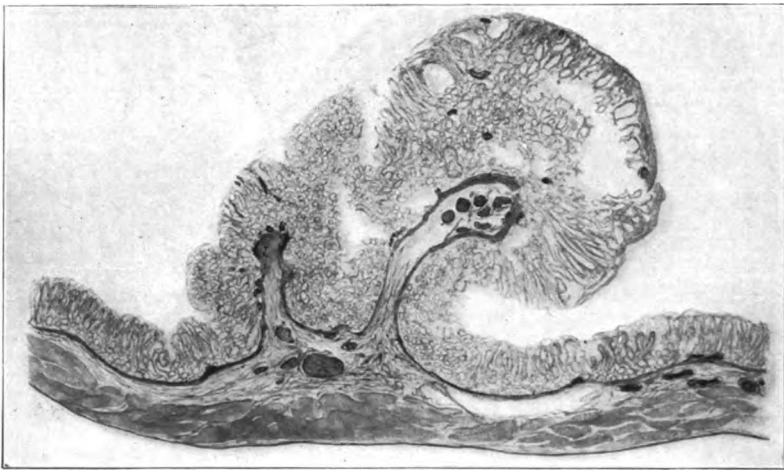
⁵ Cancer and Tumors of the Stomach, London, 1902.

It is exceedingly difficult to distinguish between large adenomas and adenocarcinomas. The microscopical picture may be quite similar, and only the subsequent course of the disease can decide the question.

Polyadenoma; Mucous Polypi (Gastritis Polyposis).—This condition is uncommon. Fenwick¹ found the frequency of occurrence to be only 0.2 per cent. Ebstein's² figures, based on 600 autopsies, are higher, 2.3 per cent.

The cause of the condition is not definitely known, but the growths seem in some way connected with chronic gastritis. It is more frequent in males, and is rare before the age of forty. Ménétrier³ found the condition frequently associated with fibromyoma of the uterus, and atheroma of the large arteries.

FIG. 49



Gastric polyp.

A number of cases have been reported in insane or epileptic patients. In these cases the disease developed early in life.⁴

In an analysis of 34 cases, Fenwick and Fenwick found the tumor solitary in 41 per cent. In the remaining 59 per cent. the number varied from 6 to 200 tumors.

The single tumors are generally located near the pylorus. The multiple ones are widely distributed over the interior of the stomach, but are more pronounced near the pylorus. The multiple tumors may

¹ Cancer and Tumors of the Stomach, J. & A. Churchill, London, 1902.

² Arch. f. Anat. u. Phys., 1864, p. 94.

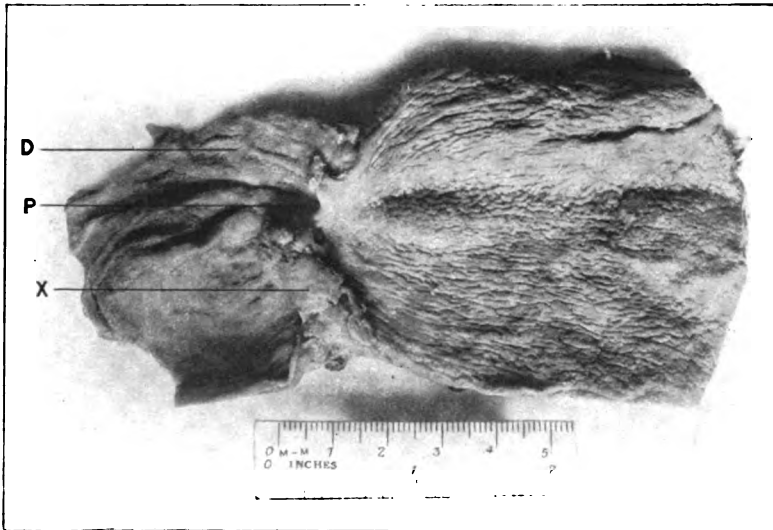
³ Arch. de Phys., 1888, ii, 32.

⁴ Stevens, Glasgow Med. Jour., 1896, p. 422; Norman, Dublin Jour. Med. Sci., 1893, p. 346.

be arranged in small groups, or in rows parallel to the long axis of the stomach. The surrounding mucous membrane may show small beginning tumors. The tumors in the stomach may be only a part of a similar condition affecting the intestinal tract.

The tumors are rounded or irregular in shape, more or less broadly pedunculated, and quite uniform in size, the multiple ones being smaller than the single variety. In color they are dirty brown or pinkish brown. They are generally covered with adherent mucus, and the surface is more or less pigmented. On pressure they exude considerable slimy mucus.

FIG. 50



Simple polyp of pylorus. About one-half of the growth was removed for microscopical sections before the picture was taken. It caused moderate obstruction of the pylorus with clinical symptoms. *D*, duodenum; *P*, pyloric valve; *X*, polyp.

Microscopically these tumors show a central stalk of connective tissue, containing bloodvessels and lymphatic. Covering this one may see the delicate prolongation of the muscularis mucosæ. Surrounding the central stalk is an exuberant growth of mucous membrane, the glands being elongated, tortuous, and dilated. In many places they form cysts and are filled with mucus. The intervening mucous membrane generally shows chronic inflammation.

Ménétrier¹ describes a more diffuse form with hyperplasia and hypertrophy of all the glands over a large area of the stomach. To this condition he applies the name "polyadenome en nappe."

¹ Les Tumeurs, *Traité de Path. Gén.*, Paris, 1899, iii, 844.

Symptoms.—These depend largely upon the location of the growth. Fenwick¹ finds that in nearly one-half of the cases in which the fundus or central portion of the stomach is affected no symptoms are observed, while in the remainder there are only symptoms of disordered digestion. Gourrand² and Quain and Beardsley³ report cases in which a mucous polyp was vomited.

A large number of the patients complain of gastric discomfort with nausea and vomiting. When situated near the pylorus, the tumor may cause some obstruction, and if pedunculated may cause intermittent obstruction and dilatation, as in the case described by Bennett.⁴ Stevens⁵ reports a case of multiple polypi in a patient subject to epileptic fits, the aura always arising in the stomach.

An unusual case of gangrenous gastritis from strangulation of a polyp in the stomach is described by McCosh.⁶ Collier⁷ reports a case of fatal intussusception of the duodenum, due to a polyp in the duodenum near the pylorus. At autopsy there was an enormous number of polyps varying in size from a pea to a pigeon's egg, scattered throughout the stomach and small intestine.

In 1908, Wegele⁸ described a case of long-standing chronic gastritis, in which at every passage of the stomach-tube a small piece of tissue was left in the eye of the tube. Microscopical examination of this tissue showed the picture of an adenoma with transition to carcinoma in some places. At operation the interior of the stomach was found covered with a large number of papillary, larger and smaller, soft polyps.

Treatment.—As the diagnosis is practically impossible in nearly every case, the treatment must be entirely symptomatic. If the symptoms become very severe, operative interference must be considered.

Other Rare Tumors.—Very rarely more unusual tumors are encountered in the stomach. Webster⁹ has reported an osteoma causing pyloric obstruction.

Myxomas and angiomas have been observed very rarely.¹⁰

Occasionally one meets with cysts in the stomach wall. They occur

¹ Loc. cit. ² Jour. de Méd., Chir., Pharm., Paris, 1790, iv, 366.

³ Trans. Path. Soc., London, 1856-7, viii, 219.

⁴ British Med. Jour., 1900, i, 241.

⁵ Glasgow Med. Jour., 1896, xlv, 422.

⁶ Annals of Surgery, 1900, ii, 630.

⁷ Trans. Path. Soc., London, 1896, p. 46.

⁸ Mittl. a. d. Grenzgeb. d. Med. u. Chir., 1908, xix, 53.

⁹ London Med. and Phys. Jour., 1827, N. S. ii, 433.

¹⁰ Hausemann, Centralbl. f. allg. Path., 1895, p. 717; Stockis, Annales de la Soc. Med.-Lég de Belge., 1905, xvi, 61.

as simple retention cysts of the gastric glands, following trauma, or as a degeneration process in various tumors. Very rarely one meets with a hydatid cyst. Ryuschi¹ has described a dermoid cyst

FOREIGN BODIES IN THE STOMACH

The accidental swallowing of small articles is not uncommon among children. Tin whistles, coins, marbles, and small toys carried in the mouth not infrequently disappear in this manner. In the majority of these instances, especially if the object is without sharp projections, it is passed through the pylorus, into the bowel, without symptoms, and eventually expelled through the rectum. Instances are on record of the uneventful passage of an opened safety-pin through the entire digestive tract.²

More rare are the cases of the lodgement of foreign bodies in the stomach. They may be of almost infinite variety, embracing hair balls, vegetable masses, gastroliths, hardware, insects, slugs, worms, leeches, lizards, and snakes. They may enter the stomach as the result of accident, insanity, or unusual or unclean habits.

Hair balls are among the most remarkable of these. They occur almost always in young girls who wear the hair long and loose, and who are usually of normal mentality, although they have acquired the habit of biting off or pulling out and swallowing hair, often unconsciously. Some have also a habit of removing and swallowing fibers from blankets, carpets, or any similar material within reach.

While often strands of hair and fibers of cloth may pass through the entire digestive tract and be discovered in the stools, still if ingested in considerable number they show a particular tendency to become matted together in the stomach, forming masses of various sizes. At first their form is globular or ovoid, and they may form a sort of ball-valve in the pyloric end of the stomach. As they increase in size, their shape becomes that of the interior of the stomach, almost completely filling the cavity, and even with projecting processes into the duodenum, and rarely into the esophagus. For this reason, the term *hair cast* is more accurate in describing the condition in the human subject. They are usually single, but there may be one or two smaller secondary masses. They are usually rather loosely woven, and of a felt-like consistency, never becoming as hard as those commonly observed in the stomachs of horses, cows, and other animals.

¹ *Adversaria Anat.*, Decas Tertia, I, "De Atheromate," p. 2; in "Opera Omnia," Amstelodami, 1737.

² B. van D. Hedges, *Med. Rec.*, March 10, 1906, lxi, 389.

The conditions produced in the stomach by the presence of a hair ball varies greatly; as a rule there are more or less dilatation, chronic inflammatory change in the mucosa, sometimes with areas of atrophy, and not uncommonly with ulceration, especially at or near the pylorus. A striking feature of many cases, however, is the remarkably normal appearance of the gastric mucosa after the presence for years of a large hair ball.

Perforation of the stomach or duodenum is not uncommonly met with. Instances are recorded of intestinal obstruction caused by small hair balls, or detached portions of larger ones. Papillomas have been found to coexist in some cases, when they were thought to be the result of the chronic irritation.

Vegetable balls are less common. They may be made up of the skins or stones of fruit, or they may result from the practice of drinking mixtures prepared from certain vegetable roots of supposed medicinal value. A case has been reported in a patient who ate not only the inside but the outside of a cocoanut.¹

Gastric concretions, although very rare, have been known in men whose desire for alcohol was so great that they would habitually drink shellac, varnish, or other resinous substances in alcoholic solution.

Hardware, of various kinds, is occasionally found in the stomachs of insane patients, jugglers, and sword swallows. The insane who are in the habit of swallowing foreign bodies are prone to select something very hard and often of considerable size. Those who give exhibitions of swallowing large hard articles are sometimes unable to recover them. The nature, number, and variety of hard articles that have been removed from a single human stomach is almost beyond belief: Nails, screws, pins, buttons, bolts, pieces of glass, false teeth, pocket knives, button hooks, and all manner of carpenter's tools have been found lodged in some portion of the stomach, usually the cardiac end, in which they form a pouch. Ulceration and perforation, with protective adhesions, are particularly common with this type of foreign body.

Living creatures also may live for a time in the human stomach, often for a sufficiently long period to be the cause of considerable gastric disturbance. Among these have been found beetles, butterflies, moths, maggots, leeches, caterpillars, worms, lizards, and frogs. The accidental swallowing of a snake has been recorded, but it is improbable that these, or any of the amphibiae, live long in the stomach, being vomited or passed by rectum. Intestinal parasites, especially *ascaris lumbricoides*, may ascend and inhabit the stomach, but usually for a short time only, as

¹ Hichens and Odgers, British Med. Jour., March 16, 1912, p. 606.

they are promptly vomited. Fenwick has collected a large series of cases in which living creatures were found in the stomach.¹

Symptoms.—Small articles usually pass through the stomach without giving any symptoms. Even those with sharp points safely pass through both stomach and bowel in a surprisingly large proportion of cases, although symptoms of perforation may be expected at any time until they are expelled. The x-ray in case the material will cast a shadow, and gives the most definite and valuable information of the progress of the foreign body in its course through the stomach and intestines. It is often of the greatest aid also in making a negative diagnosis in those supposed cases of swallowed foreign body which are the result of an active imagination in a child, or a hasty conclusion on the part of a terrified parent.

FIG. 51



Hair ball removed from stomach of a woman, aged twenty years. (From the Pathological Museum, Columbia University, New York.)

The development of a hair ball in the stomach usually occurs in patients who give no history whatever suggesting the cause of the condition. It may be attended by remarkably few symptoms. The size which such a mass can attain in certain cases without noticeably interfering with the gastric functions is surprising, but sooner or later there is usually a more or less prolonged period of indefinite digestive disturbance which progresses to definite pain and vomiting.

The pain is most common after meals, and may be felt at first throughout the abdomen, later being more localized in the epigastrium or left hypochondrium, and being increased by pressure. It may be accompanied by flatulency, distention, and nausea. The vomiting usually

¹ W. Soltau Fenwick, *Dyspepsia*, 1910, p. 287.

occurs at the time of greatest pain, sometimes regularly after every meal. The vomitus when typical is small in amount, acid, and may contain bile or possibly blood, but almost never hair. Other less constant symptoms are coated tongue, foul breath, and alternating diarrhea and constipation, and a variable degree of anemia. The appetite is not always affected, and loss of weight and strength do not appear until later. If ulceration has taken place, the picture may be modified at any time by the symptoms of hemorrhage or perforation. Gastric tetany has occurred.

The feature of the physical examination is the tumor. When the patient is in the dorsal position the greater part is usually to the left of the median line, in the epigastrium, and left hypochondrium. It may be globular, ovoid, or assume quite accurately the size and shape of the stomach, and there may be one or two smaller secondary masses. Its mobility is a distinguishing characteristic in cases without adhesions. It is smooth, quite hard, feels close to the abdominal wall, and gas may be felt during palpation. The presence of tenderness depends chiefly upon ulceration.

In cases in which the habit is not corrected or the mass is not removed by operation, the condition is steadily progressive. A case has been recorded of which the duration was twenty-two years. Death occurs most often from inanition, to which the diarrhea in some cases contributes, or from perforation.

The chief difficulty in diagnosis is that the condition is so rare that the physician seldom has it in mind. Some of the conditions for which hair ball may be mistaken are gastric cancer, displaced kidney or spleen, omental tumor, and fecal impaction. Retroperitoneal sarcoma, pancreatic cysts, and tuberculous glands are less apt to be considered because they are usually less mobile. The chief features of hair balls which are useful in diagnosis are: their occurrence almost always in girls who wear the hair long, the long duration of symptoms, and the mobility and smoothness of the tumor. The relationship of the tumor to the stomach may be shown by careful inflation of that organ, but better still by the *x*-ray after the ingestion of bismuth.

Vegetable balls are even more rare than hair balls, seldom reach such large size, and occur in older patients of both sexes from whom it is less difficult to obtain a history of the causative habit or mental condition. The detachment of fragments and their expulsion per rectum are more frequent.

The symptoms in cases of hardware in the stomach are seldom apt to be characteristic, and their entire absence is often remarkable. The type of patient, history, and *x*-ray are the most usual aids to correct diagnosis.

Living creatures make their entrance into the stomach often as larvæ or ova which have been deposited on food, either after its preparation, or on uncooked articles, or from eating improperly prepared salads or drinking impure or stagnant water. Fenwick cites a case which resulted from the habitual eating of clay considered sacred by a superstitious young woman. Instances of these conditions are more common in tropical than in temperate climates. Their presence is usually made known by their discovery in the feces or vomitus. They produce indefinite gastric or intestinal symptoms, unusual sensations, or vomiting with or without persistent diarrhea. Fever is not uncommon, and in children there may be convulsions. Those hysterical patients who frequently describe sensations, which they are convinced are caused by a living object in the stomach, as well as those who deliberately intend to give a false impression by adding insects or other creatures to specimens of feces or vomitus, must be sharply differentiated from those who actually harbor such objects.

Treatment.—In cases among children in whom small foreign bodies have entered the stomach after having been accidentally swallowed, it is, as a rule, unwise to administer emetics or cathartics. A diet of mashed potatoes, with cream and butter, for a few days may have the effect of surrounding the article with a starchy coating which facilitates its safe passage through the intestine. Oil enemas may be used if necessary to secure bowel movements. This class of cases is best treated conservatively, especially in infants and very young children, in whom the risk of operation is relatively great. The patients should be carefully watched, if possible with the assistance of the x-ray, when surgical intervention may be resorted to without delay in case symptoms of obstruction or perforation arise.

When gastric or intestinal symptoms coexist with the habit of swallowing hair, but when no tumor can be felt, detection and correction of the habit may be followed by a complete disappearance of the symptoms. But in the case of hair balls, vegetable balls, or gastric concretions of sufficient size to be palpated or of hardware lodged in the stomach, the only treatment is operation. Attempts at removal by means of emetics or cathartics are attended by too much risk and too little chance of success to be permissible.

Prophylaxis is of prime importance when habits resulting in the ingestion of larvæ or ova are detected. A thorough catharsis may result in the expulsion of all of the insects or animals, but when they are more resistant or numerous, thymol, santonin, or other anthelmintic is necessary.

CHAPTER IX

TUBERCULOSIS AND SYPHILIS OF THE STOMACH

TUBERCULOSIS OF THE STOMACH

Tuberculosis of the stomach is a disease of comparative rarity, as it is present in but 0.5 per cent. of all autopsies, and in but 2.3 per cent. of the post mortems done on those dead from tubercular disease. It is somewhat more common in acute miliary tuberculosis than in the chronic forms of infection. Difficulty in drawing conclusions from the ordinary postmortem statistics as to its frequency lies in the fact that unless microscopical examination shows a typical tubercular picture, or unless tubercle bacilli can be demonstrated in the tissues, there is always a doubt whether the lesion is tuberculous or not. Not infrequently do those affected with phthisis suffer from the ordinary form of gastric or duodenal ulceration, so that without microscopical or bacterial examination it cannot be assumed that a particular ulceration found at autopsy in a tuberculous patient is a secondary infection resulting from the pulmonary complaint, or an independent and coexisting ailment.

Mode of Infection.—1. It is plausible to assume that infection of the stomach by the tubercle bacilli would most readily occur by the direct contact of the mucous membrane by infected sputum arising from tubercular disease of the upper air passages, or by food that has been contaminated by tuberculous dust. It is obvious, however, that this mode of infection is not as frequent as one would expect. The peristaltic power of the stomach to empty itself is a far more potent protection against tubercular infection than is the bactericidal affect of the gastric juice, as it requires twelve to eighteen hours' immersion in this secretion to destroy the bacillus. The poverty of the stomach in lymph follicles as distinguished from the abundance of this tissue in the intestinal tract is an additional reason why the stomach should be much less frequently affected than the intestine by tubercular disease.

2. The bacilli may infect the stomach wall through lymphatic vessels or through the contiguity of tuberculous perigastric glands. In this manner the stomach is often involved as a secondary infection

to tuberculous lesions in the intestine or peritoneum, even though the lungs be free from all tubercular taint.

In a case reported by Clayton and Wilkinson,¹ there were three caseous glands in the neighborhood of the ulcer, one of which was adherent to its base and showed microscopical communication with it. A similar case is reported by Besnier in which a man without pulmonary complaint was found to have tuberculosis of his abdominal glands, one of which had suppurated and had perforated into the posterior wall of the stomach.

3. Arloing believes that infection through the blood current is the most usual method, as he has been able to produce tuberculous gastric and duodenal ulceration by injecting bacilli into the blood. This theory is further supported by the fact that these tuberculous ulcerations are more frequently observed in acute disseminated tuberculosis than in the chronic forms of the disease in which the process is more localized.

It is a rarity to find tubercular disease of the stomach as a primary infection, as in almost all instances the gastric disorder is secondary to or is associated with tubercular disease of the lungs and upper air passages or of the intestines and peritoneal tuberculosis with involvement of the perigastric glands. The symptoms, therefore, of gastric origin are frequently so intermingled with those of the primary ailment that the clinical course of the disease is often involved and obscure.

Nor can it be affirmed that because a patient with tuberculosis complains of his digestion that the cause for his discomfort lies in an organic involvement of the stomach by any tuberculous process. Tuberculous patients are apt to suffer from functional derangements of their digestion either by reason of failure of their general strength or because the stomach has been overloaded by food or by large quantities of milk and cream. In other instances, chronic gastritis with acute exacerbations may occur, due in all probability to the toxins generated by the primary disease. There are many cases of acute phthisis which begin their clinical course by excessive vomiting, apparently of toxic origin. In more advanced cases it is not unusual for atrophy of the mucous membrane of the stomach to occur. Tuberculous patients who are enteroptotic, regularly suffer from the symptoms of this ailment in an aggravated form whenever they lose flesh and strength by reason of their pulmonary complaint. In many cases one of the above-mentioned types of dyspepsia may constitute for a time the sole symptom of the pulmonary disease, amending or altering its type as the pulmonary disease progresses, and gradually subsiding as the tuberculous process becomes arrested. These forms of indigestion must always be excluded

¹ Arch. Int. Med. April, 1905, p. 825.

before we can assume that the dyspeptic complaints of phthisis are due to an actual infection of the stomach by a tuberculous process.

The tuberculin test is rarely of service, owing to the presence of other obvious tuberculous lesions in other parts of the body. The ophthalmic test and the cutaneous reaction test of von Pirquet have long since been abandoned.

Forms.—Aside from the deposition in the stomach wall of miliary tubercles that occur with acute miliary tuberculosis, and which are of anatomical rather than of clinical interest, tuberculosis of the stomach occurs in two principal forms:

1. Tuberculous ulcer.
2. Tuberculous tumors with pyloric stenosis.

Tuberculous Ulcers.—The ulcers vary in size from that of a dime to large irregular excavations $1\frac{1}{2}$ or 2 inches in diameter. In the case of Clayton and Wilkinson, a large single ulcer was found surrounding the esophageal opening, 9 by $4\frac{1}{2}$ centimeters in size. Any portions of the stomach may be involved, although the lesser curvature and the pyloric region are its favorite seats. The edges are usually raised above the surrounding tissue, are thickened and nodulated, and often present a scalloped appearance. As the disease process usually begins in the submucosa, it may happen that the edges are deeply undermined. The base may be grayish or yellowish, and presents a granular aspect from irregular deposits of tuberculous tissue. Such ulcerations may be either single or multiple. In Ellis'¹ case 8 ulcers were found. The peritoneum covering the base of the ulcer almost always exhibits a few miliary tubercles, and is not infrequently adherent to neighboring parts. The perigastric glands seem regularly to be involved, either secondary to the ulcer itself, as is the case when infection has occurred through the swallowing of sputum or contaminated food, or the inflammation of the glands may be secondary to tubercular disease of the peritoneum or intestines, and through contiguity of tissue may cause a further involvement of the stomach.

Perforation into neighboring parts may ensue. The duodenum has been known to be perforated, forming a gastroduodenal fistula with temporary relief from the symptoms of pyloric stenosis. Gastrocolic fistulas have also been noted.

SYMPTOMS OF TUBERCULOUS ULCER.—In the majority of cases the symptoms of the ulcer itself are so obscured by those of the original tubercular disease that the diagnosis is unsuspected. Of the gastric symptoms proper, pain, hemorrhage, and perforation are the most important.

¹ New York Med. Jour., March 12, 1910.

Pain.—Pain is usually a well-marked symptom and resembles that of the ordinary gastric ulcer in having a fixed relation to the taking of meals. The relief afforded by eating is, however, less noticeable and of shorter duration than is the case with the simple ulcer. When the peritoneal coat is involved, pain may be more constant and severe and is associated with well-marked tenderness in the region of the stomach. Rigidity of the upper abdominal wall is indicative of such a peritoneal involvement. As compared with the ordinary form of ulcer, the pain of tuberculous ulceration is more constant, more severe, and is not marked by the intermissions so commonly observed in the non-tubercular form. The distress, furthermore, is apt to occur sooner after the taking of food than is usually the case in the non-tubercular form of ulceration.

Hemorrhage.—Hemorrhage may occur both in the visible and occult form, but, as a rule, profuse hemorrhages are rare.

Perforation.—Perforation is not an uncommon event, and may be the first symptom that indicates the presence of the ulcer. If the patient be in a fair condition of health, the symptoms are those of perforation in its classical and typical form; but should the patient be much debilitated by the tubercular disease, the initial symptoms of shock are less marked; but the symptoms of acute peritonitis, nevertheless, develop and death rapidly supervenes.

Tuberculous Pyloric Stenosis.—The deposition of tuberculous tissue of the hyperplasia type in the neighborhood of the pylorus may result in the formation of a tumor in this situation obstructing the pyloric orifice. As a rule, this form of tuberculous deposition is rich in connective tissue, shows but few giant cells, and contains but few bacilli, thus indicating an attenuated infective process. In some instances this attenuation is so marked that microscopical examination fails to reveal any apparent evidence of tubercular nature.

In a case reported by Chalié,¹ a thickening of the pylorus existed without any specific tubercular change, and yet inoculations of the guinea-pigs with scraps of tissue from this neoproduction resulted in the death of the animals from tuberculosis. It is possible that many cases of fibrous stenosis of the pylorus might be found in this way to be of tubercular origin. Chalié states that the structure of the stenosis in his case resembled so closely the lesions encountered in stenosis of the pylorus in the newly born that it suggested a possible similar origin in the latter cases.

¹ Tumeur inflammatoire sténosante du pylore d'origine tuberculeuse (Rapports de l'inflammation avec l'hypertrophie tumeuruse du tissu musculaire lisse), J. Chalié and L. Nové Josserand, Lyon Chirurgical, 1911, Tome vi, No. 4, pp. 389-412.

SYMPTOMS.—The symptoms of tuberculous pyloric stenosis closely resemble those of malignant origin. Food-stasis is commonly observed, as is shown by the passage of the tube in the fasting state and by the vomiting of the patient from time to time of food that has been lying in the stomach for many hours. The pain and discomfort are those ordinarily observed in pyloric stenosis, and do not serve to differentiate the particular type of contracture.

Diagnosis.—Gastric analysis is practically identical with that observed in carcinoma of the pylorus. The fasting stomach contains evidence of food remains, with or without lactic acid and lactic acid bacilli. Test breakfast shows a diminution or absence of free and combined hydrochloric acid and the presence of lactic acid in appreciable amounts. Microscopically the Oppler-Boas bacilli are not infrequent. Physical examination shows a hard, movable, elongated, and slightly tender tumor below the right costal margin in the position of the pylorus. A well-marked pyloric squirt may be audible through the stethoscope. Should the peritoneum covering the tumor be the seat of tuberculosis, marked tenderness and rigidity will be present.

Prognosis.—The prognosis depends as much if not more upon the extent of the other tuberculous lesions of the body than upon those in the stomach. Recovery is possible, but the chances for it are slight.

Treatment.—Medical treatment is not of much avail in producing any direct effect upon the healing of the lesion, although some degree of relief to the distressing symptoms may result from careful management of the case. The dietetic rules are those of ulcer generally. Lavage may be of service if food retention should occur from narrowing of the pyloric orifice. The results of medical treatment are, however, most disappointing, as a rule.

Operative interference is not generally advisable, unless to relieve pyloric obstruction. If the diseased process at operation seems to be limited to the pylorus, and if the other tuberculous lesions are not such as to obviously limit the expectation of life, partial gastrectomy may be performed.

Excision of tuberculous ulcerations at parts of the stomach other than the pyloric portion is an operation not lightly to be advised, although such a process may be justifiable if the process is limited to the neighborhood of the ulcer, and if the other tuberculous lesions are not sufficiently advanced to threaten life in the near future.

Of 20 reported cases in which operations were done for tubercular disease of the stomach, Deaver could trace the end results in 18. Of the 18 patients, 5 died from the operation. Of the remaining 13, 8 were traced to their death, which occurred at an average period of eight months after the operation. One patient lived for three and a half

years, finally succumbing to an abscess of the liver. In the remaining four the period of time that had elapsed after the operation, at the time of their report, was too short to be conclusive.

SYPHILIS OF THE STOMACH

Syphilis of the stomach occurs as a rare localization of the visceral form of late syphilis, either hereditary or acquired. Chiara found specific lesions present in the stomach in but 3 out of 242 autopsies performed on syphilitic subjects. Of these 1 was hereditary, 2 were acquired.

It is estimated that there are on record in the neighborhood of 50 cases of gastric syphilis. It is probable, however, that the disease is much more frequent than this, and that it is our failure to correctly diagnose the ailment that accounts for its supposed rarity. It must be remembered, however, that ordinary ulceration of the stomach may occur in syphilitic persons with the same frequency as it does in those without this constitutional taint. Again, it must be remembered that an antisyphilitic treatment will favorably influence non-specific affections in syphilitic patients by improving their general health. Nor must it be forgotten that a rigid course of mercury and potassium iodide may so derange the digestion as to produce symptoms either of chronic gastritis or of functional dyspepsia.

Allen A. Jones has called attention to the frequency of neuralgic pains in the region of the stomach in syphilitic patients. These he considers to be of functional nature, due to the disturbances in the general condition of health. They are usually amenable to antispecific treatment.

Pathology.—The essential lesion consists of the deposition in the submucosa of gumma or of gummatous tissue either in a circumscribed or in a diffuse form. Giant cells with peripheral position of the nuclei (Langhans type of giant cells) may be found throughout the diseased tissue. Such an infiltration may extend secondarily to involve the mucus or the outer layers of the stomach wall. Surrounding the gumma is found a zone of granulation or fibrous tissue depending on the age of the lesion, which may be regarded as due to reparative reaction of the adjacent tissues. In other cases the gumma shows a gradual gradation into normal gastric tissue and has no fixed line of demarcation. Gummas are apt to become necrotic in the centre and may rupture through the surface. In the gumma there almost regularly occur concentric deposits of gummatous tissue around and in the walls of the small bloodvessels, so that an obliterative form of endarteritis

results, which by diminishing the normal blood supply still further increases the tendency to necrosis. In other instances the necrotic portion may become absorbed and replaced by a mass of cicatricial tissue. In other cases the gummatous deposit does not undergo necrosis, but may either undergo resolution or may become converted into a diffuse mass of fibrous tissue. The absence of the spirochete is not to be considered an evidence against the specific nature of the affection.

Syphilis of the stomach occurs in four principal forms:

1. Syphilitic ulcer.
2. Syphilitic tumor.
3. Syphilitic pyloric stenosis.
4. Syphilitic cirrhosis.

In all these forms, and occasionally in syphilitic patients who do not present one of the above-mentioned types of lesion, we are apt to have a well-marked catarrhal gastritis, evidently due to the effect of syphilitic toxins.

Secondary and associated lesions are often found in other parts of the body. Syphilitic tumors, ulcers, and stenosis may be found in the jejunum, sigmoid, and rectum, and in the esophagus. Gummas are often found in the liver, occasionally attaining considerable size. Their favorite seat appears to be in the left lobe or in the neighborhood of the suspensory ligament, at a point where the liver undergoes a certain degree of traumatism by traction of the ligament at this point.

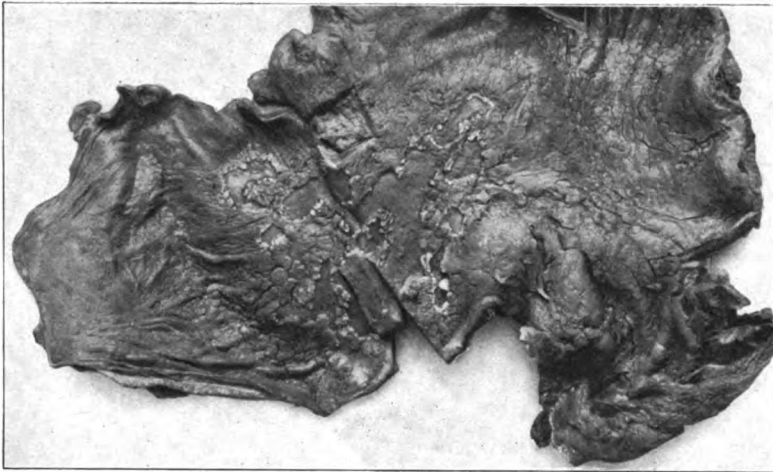
Syphilitic Ulcer.—Syphilitic ulcers may be either solitary or multiple, and occur in any part of the stomach, although they are somewhat more frequent in the region of the pylorus and lesser curvature. They vary very considerably in size and may attain considerable proportions. The ulcers may be superficial, extending only partially through the mucosa, or they present a deep excavated appearance, often having a serpiginous outline. Syphilitic ulcers are regularly the result of the breaking down of gummatous tissue, which has either become necrotic or which has become devitalized by reason of the diminished blood supply through the partially obliterated bloodvessels and has undergone erosion by the peptic power of the gastric juice.

Should specific endarteritis occur with but scanty submucous deposits of gummatous tissue, peptic erosion may occur of the area rendered thus anemic from lack of sufficient blood supply to resist the corrosive action of the gastric juice. An ulcer formed in this way does not present the usual appearance of syphilitic ulcer, as its walls and base are not formed of necrosing gummatous tissue, but may be quite indistinguishable from the ordinary form of gastric ulcer, although microscopical examination may show the characteristic of syphilitic endarteritis so that there is no doubt of the specific origin of the ulcer.

Symptoms.—The symptoms of syphilitic ulcer do not vary materially from those of the non-specific form. Hemorrhage or perforation may occur and either complication may be the first indication of the gastric disease.

In one of the writer's patients, a young man was taken in the street with such severe hematemesis that he was brought by ambulance into the hospital in an exsanguinated condition, and utterly unable, by reason of his weakness, to give any facts of his past history. The hemorrhages continued in spite of all treatment and he died eight hours after his admission.

FIG. 52



Syphilitic ulcer of the stomach. The history of this case is given in full in the text.

The mucous membrane of the lesser curvature was the seat of an extensive serpiginous ulceration involving an area approximately 6 by 5 c.m. The edges were irregularly sinuous, slightly raised but not indurated. The floor was composed of the submucosa, was clean and not covered by slough. About 2 cm. from the left border of the cardiac orifice just within the margin of the ulcer were seen the eroded ends of two vessels which projected slightly above the surface of the ulcer. About 3 cm. from the pyloric ring along the greater curvature is a small polypoid outgrowth, adjacent to which was a superficial irregular ulcer 2 cm. in diameter having the general characteristic of the larger one just described.

The wall of the stomach from the pyloric ring to beyond the limits of the ulcer was markedly thickened by the replacement of the sub-

mucosa by firm, whitish, infiltrating tissue. There were multiple gummas in the liver. The mesenteric lymph nodes showed circular areas of firm whitish tissue. A detailed account of the microscopical findings can be found in Pappenheimer's¹ report of the case.

Diagnosis.—The diagnosis of syphilitic ulcer cannot be made on the evidence only of the gastric symptoms presented, but it is to be surmised when the symptoms of ulceration of the stomach are complained of by a luetic patient who has obtained no relief by the ordinary forms of treatment for his ulcer. This suspicion is strengthened to the point almost of certainty should rapid and lasting recovery follow an antisyphilitic course of treatment.

Syphilitic Tumors.—Every gumma is a tumor, but the term tumor is clinically applied only to those cases in which the growth is of sufficient size to be detected by palpation. Ordinarily gummas are not large enough to be easily detected, but there are cases in which they attain the size of an egg, or even larger than this, and may be easily detected by physical examination. They may be single or multiple, occurring in parts of the body other than that of the pylorus. They give no characteristic symptoms. Pain in the stomach after eating, with occasional vomiting, may be complained of, together with considerable loss of flesh and strength; but these are the symptoms common to a great number of gastric disorders, and are not in themselves of diagnostic value. A firm nodular resistance is, however, felt over some portion of the stomach; which may so closely resemble cancer that a differential diagnosis is absolutely impossible. In one of Einhorn's² cases the tumor was of the size of a goose egg.

The difficulty in making a diagnosis between syphilitic tumor and cancer is further increased by similarity in the gastric analysis, for in the majority of syphilitic tumors of the stomach hydrochloric acid, both free and combined, is absent, and lactic acid may be present in small amounts.

This was the gastric analysis found in the cases reported by Curtis,³ but it is not the invariable rule, for in one case reported by Einhorn,⁴ lactic acid was not present, although hydrochloric acid was absent, while in a second case by Einhorn,⁵ the total acidity was 40 and free hydrochloric acid was present.

The diagnosis is to be suspected in every case of tumor of the stomach

¹ Medical and Surgical Report of Bellevue and Allied Hospitals, 1905-6, ii, 219.

² Philadelphia Med. Jour., February 3, 1900, p. 264.

³ Jour. Amer. Med. Assoc., April 10, 1909.

⁴ Philadelphia Med. Jour., February 3, 1900.

⁵ Dermatologische Zeitsch., 1900, p. 450.

that occurs in syphilitic subjects, especially if other evidences of the luetic poison such as gummas of the liver or lesions in the bones can be detected.

Syphilitic Pyloric Stenosis.—Stenosis of the pylorus in constitutional syphilis may occur in a variety of ways. There may be a thickening of the wall of the pyloric canal, either by gumma or by dense fibrous tissue, which represents one of the end results of the syphilitic infiltration. In other cases the healing of an ulcer in this situation may be followed by a cicatricial contraction of the orifice. The symptoms of pyloric stenosis thus induced do not differ from those of pyloric stenosis in general.

The fasting stomach contains residual food, indicative of a grave motor error. Gastric analysis has not been made in a sufficient number of cases to enable us to formulate any opinion upon its value as a diagnostic aid.

In two cases reported by Einhorn the total acidity was high and free hydrochloric acid was present. The differential diagnosis cannot be positively made from benign stenosis or from cancerous tumor of the pylorus.

Cirrhosis of the Stomach of Syphilitic Origin.—It is not uncommon that gummatous infiltration shows a tendency to pass into a condition of fibroid induration which may be localized in the neighborhood of the pylorus leading to pyloric stenosis, or which may be diffuse throughout the stomach wall, so that the stomach becomes contracted, its walls thickened and fibrous, the appearance being apparently identical with that of the non-specific form of cirrhosis of the stomach, so that a differentiation between them, save by most careful microscopical search, cannot be made.

Such a case was reported by Hemmeter and Stokes,¹ The patient was a young man, aged twenty-four years, who gave the history that two years previously he had contracted syphilis. Gastric analysis showed absence of hydrochloric acid. The stomach was appreciably diminished in size. Death occurred after operation for the relief of pyloric stenosis. Autopsy showed the stomach wall thickened, especially in the region of the pylorus, where the opening would barely admit a lead-pencil. A number of cases have been reported of this form of syphilitic fibrosis in which operations were done for pyloric stenosis.

It does not seem possible that a differential diagnosis can be made clinically between benign fibrosis of the stomach (the so-called cirrhosis ventriculi), fibroid cancer of the stomach wall, and this form of syphilitic fibrosis.

¹ Arch. f. Verdauungskrank., 1901, vii.

Treatment.—The treatment of gastric syphilis is that ordinarily employed in tertiary lesions. A thorough course of iodide and mercury is indicated and should be continued so long as any improvement follows the treatment. Injections of salvarsan may also be given if the diagnosis is established. A case of diffuse syphilitic infiltration from the stomach of cirrhotic type is reported by Hausmann¹ as having been cured by an injection of salvarsan.

Fibroid induration of pyloric ulceration or gummas that result in permanent stenosis may be treated surgically by exsection or gastro-jejunosomy.

¹ Münch. med. Woch., 1911, No. 10, p. 511.

CHAPTER X

ATONY OF THE STOMACH

General Considerations.—Normal Tonicity of Stomach.—Atony of the stomach is the condition in which the stomach wall has lost its tonicity. A few words of explanation of what is meant by tonicity may be necessary. Three forms of muscular contraction are observed in the normal stomach. The first form consists in a concentric drawing together of the stomach so as to adapt itself to the volume of its contents and to maintain a certain uniform pressure upon them. This concentric contraction of the stomach on its contents is often spoken of as the perisystole of the organ, and the diminution of this motor function results in what we term "atony." Tonus is essentially the function of the central portion and fundus of the stomach. The second form of muscular contraction is a vermicular series of constricting rings running in the direction of the longitudinal axis from left to right. This is called peristalsis and its object is to propel the contents of the stomach from the fundus toward the pylorus. Peristalsis is especially the function of the prepyloric portion of the stomach. These two motor functions, tonus and peristalsis, while distinct from each other, are to a certain extent correlated so that both may be affected at the same time, although one may be impaired and not the other.

The third form of motor activity of the stomach is shown by the alternate contraction and relaxation of the pyloric sphincter regulating the outgo of food. The pylorus remains tonically closed even against recurring pressure when food is taken. The appearance of acid at the pylorus causes the sphincter to relax so that peristalsis is able to expel some of the acid chyme into the duodenum. The acid in the duodenum at once closes the sphincter until the acid has been more or less completely neutralized by the duodenal secretions. As neutralization proceeds the duodenal stimulus causing the closure of the pylorus becomes weakened so that the acid in the stomach side of the sphincter is again able to produce relaxation and allow of the expulsion of more chyme. Beside this acid control of the sphincter, the pylorus exerts a selective control over what food particles may be allowed to pass—coarse food in large lumps is regularly retained in the stomach longer than digestible food finely comminuted.

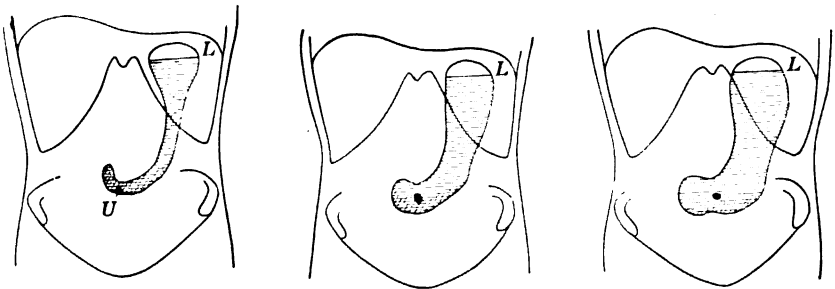
When food enters the normal stomach it does not drop into the most dependent portion as one would expect, but forms a column of about

two-thirds the height of the stomach. As more and more food is added the width of this column increases, but not the height, the upper limit being maintained whether the volume be 40 c.c. or 400 c.c. The reason for this is that the normal tonicity of the stomach wall exerts a concentric pressure on the contents, holding them in a tubular form. The greater curvature is but little depressed as the stomach is gradually filled. Above the column of food is an air chamber which is quite constant.

When atony is present this close adaptation of the stomach to its contents no longer occurs, but food drops into the most dependent portion of the stomach, lying there more or less transversely and sagging the stomach downward, so that the lower curvature may be 1 or 2 inches below the umbilicus. As more food is taken the greater curvature sinks more and more deeply, while the upper limit of the contents rises only to a slight extent. The air space is much larger than normal and assumes roughly a cylindrical form with a bulbous upper extremity.

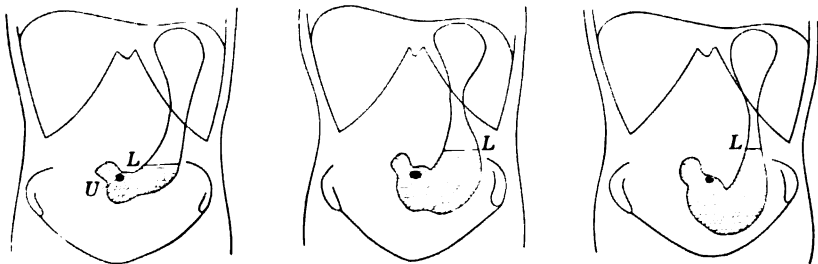
The difference in the filling of the normal and atonic stomachs may be shown by the accompanying diagrams.

FIG. 53



Filling of the normal stomach. U, umbilicus; L, upper limit of gastric contents. (Hertz.)

FIG. 54



Filling of the atonic stomach. U, umbilicus; L, upper limit of gastric contents. (Hertz.)

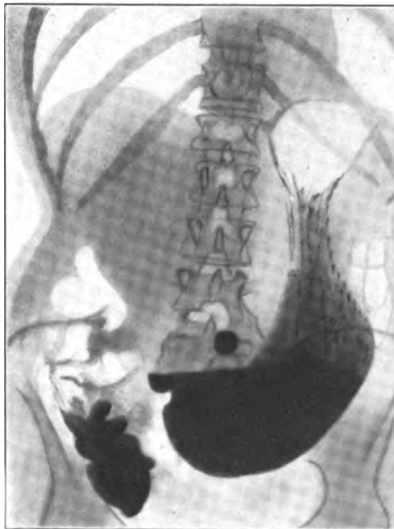
PLATE VIII

Fig. 1



Filling of the Normal Stomach. (Radiologist, Dr. Cole.)

Fig. 2



Filling of the Atonic Stomach. (Radiologist, Dr. Leaming.)

Every atony is, in its inception, a lack of tonus affecting chiefly the fundus portion of the stomach. When thus limited the peristaltic function may be quite unimpaired, so that the stomach will empty itself within proper time limits. In these cases examination by means of test meals may not reveal the least retardation in the expulsion of food from the stomach, although well-marked atony may be demonstrated by the *x*-ray.

When the process becomes more marked and more generalized, the peristaltic function becomes impaired with a resulting tardiness in the onward propulsion of food into the duodenum. This delayed exit of food may be clearly shown by the presence of test meals in the stomach after they should have been expelled, but although normal time limits may have been passed, the error is never so grave as to lead to what we might call food-stasis. The onward current may be slow, but it is certain. The stomach is regularly and invariably empty in the morning fasting state in uncomplicated cases of atony. This test alone differentiates distinctly between atony and pyloric stenosis.

Occurrence.—Atony is one of the commonest causes for dyspepsia and occurs in the writer's experience in one of every seven patients in private practice complaining of their digestion. In hospital cases the ailment is probably less than half as frequent.

Sex.—There seems to be no greater liability to the affection in women than in men, although reasoning from the etiology of the disease women would seem to be peculiarly predisposed to the ailment. In the writer's cases 39 per cent. were observed in men and 61 per cent. in women, but the number of women applying for treatment was just about in this proportion, so that the difference in frequency in the two sexes is not as great as it really would appear to be.

Age.—The functional disorder is rare in childhood, but occurs evenly distributed through the adult life if we class men and women together. In both men and women atony occurred:

Between 10 and 20 years	6 per cent.
Between 20 and 30 years	29 per cent.
Between 30 and 40 years	26 per cent.
Between 40 and 50 years	26 per cent.
Between 50 and 60 years	12 per cent.
Between 60 and 70 years	1 per cent.
	<hr/>
	100 per cent.

When we separate the men from the women we find that in women the disorder occurs at a far earlier period of life than in men.

In 100 cases of atony in women there occurred:

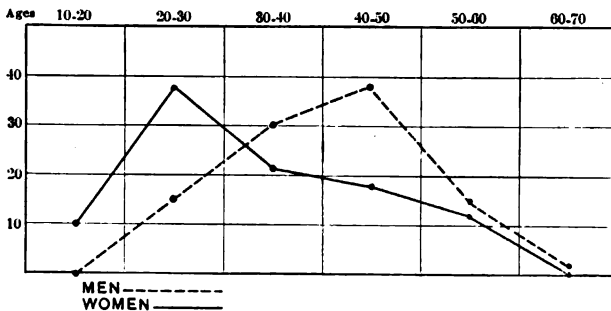
Between 10 and 20 years	10 cases
Between 20 and 30 years	38 cases
Between 30 and 40 years	23 cases
Between 40 and 50 years	18 cases
Between 50 and 60 years	11 cases
Between 60 and 70 years	0 cases
	<hr/>
	100 cases

Of 100 cases of atony in men there occurred:

Between 10 and 20 years	0 cases
Between 20 and 30 years	15 cases
Between 30 and 40 years	30 cases
Between 40 and 50 years	38 cases
Between 50 and 60 years	15 cases
Between 60 and 70 years	2 cases
	<hr/>
	100 cases

The differences in the ages at which the disorder is manifest is shown by the accompanying table:

FIG. 55



The period of greatest liability in men to gastric atony is that of the gravest responsibilities of life. The cause for the earlier appearance of the disorder in women is more difficult to understand. Many of the writer's cases occurred in nervous delicate women, socially active, living on their nerves, often with the history of rapid child-bearing and frequent pelvic operations.

Etiology.—Atony is a functional disorder due to lack of tonus of the stomach wall that may arise from general, local, or from reflex causes.

General Causes.—Every neurasthenic is predisposed to atony by reason of his nervous condition. Neurasthenia, irrespective of its

duration, is the underlying factor that induces the gastric disorder in the very great majority of instances. The nervous weakness may be congenital or acquired.

In 25 per cent. of the writer's cases there were the stigmas of the so-called congenital neurasthenia described by Stiller, the outward signs of which are shown by what is termed the enteroptotic habitus. The costal angle is sharp, measuring less than 50° to 55° ; the margins of the ribs pass more vertically downward than in normal individuals. The tenth rib is frequently unattached, the thorax is long and slender, the intercostal spaces broad and sunken. The patient is almost invariably of delicate frame and of spare habit. Visceral ptoses can be easily demonstrated. In these patients with the enteroptotic habit gastro-intestinal atony is an inherent part of a general constitutional weakness and is liable to make itself manifest by symptoms of indigestion whenever the patient runs down from any cause whatever. These are the patients who are constitutionally unable to withstand the storm and stress of daily life and who habitually have indigestion unless life is made easy for them in every possible way. This constitutional anomaly should be suspected whenever a patient, especially if thin and undernourished, complains of "having a delicate stomach" as long as he can remember.

Acquired neurathenic or psychasthenic states may follow any variety of nervous or physical strain, prolonged or temporary. Transient atony may follow sudden physical or nervous crisis, such as operations, sudden calamities, acute illness, overfatigue, sexual excesses, or prolonged lack of sleep. The removal of the exciting cause may be followed by a more or less rapid restitution, although in many cases atony induced by temporary causes may continue for weeks or may even become permanent.

Chronic Exhausting Diseases.—Atony is a regular accompaniment of all chronic exhausting diseases, of which tuberculosis and diabetes furnish perhaps the most typical examples. The excessive quantities of milk or of water taken in these two ailments further increase the degree of the atony.

Almost all patients with chlorosis show atony of the stomach of greater or less degree.

Local Causes.—The local causes for atony are not so numerous as are the general causes for nervous depreciation. Excesses of eating and drinking may be followed by the symptoms of the ailment. A very frequent cause is the habit of drinking water to excess, especially at meals, mechanically overloading the stomach by excessive weight and bulk of its contents. Physicians should be more careful and discriminating than they are in advising or allowing such a "water cure."

Many people are obsessed with the idea that the more they drink the healthier they are, and in consequence fill the stomach with water at the rate of a pound a pint at every possible opportunity. When the water is taken iced, the greater is the damage done.

Excessive quantities of food, or food improperly masticated, containing coarse indigestible lumps, are often retained in the stomach for an abnormal period of time through a selective action of the pylorus. This is a well-known cause for acute indigestion, and occurs frequently enough in the experience of everyone. When these dietetic errors are constantly repeated gastric atony may result, although cases thus induced are less frequently encountered than one would be led to suppose. Atony is relatively less common in hospital and dispensary patients who habitually ill treat their stomachs than in private patients who are careful of what they eat.

Atony is a common sequel to any form of acute gastritis and often is the means of protracting convalescence long after the acute inflammatory symptoms have subsided. With chronic gastritis atony does not appear unless due to other causes than the gastric catarrh.

Reflex Causes.—Reflex causes for atony may originate from irritative lesions anywhere within the abdominal cavity. Tumors of the ovary or spleen, epigastric hernia, mesenteric cysts, and renal calculus have all been proved to stand in a casual relationship to the disorder. Lesions of the gall-bladder and gallstones are the most frequent of these causes. Acute cholecystitis is frequently accompanied by symptoms of gastric atony, and these may be so pronounced as to overshadow those due to the gall-bladder infection and to thus obscure the diagnosis.

Appendicitis does not seem to be a frequent cause for atony, unless the appendicular disease occurs in a patient who has the enteroptotic habit and visceral ptosis. In such a subject atony during chronic appendicitis is liable to occur.

Irritative lesions of the pelvic organs in both sexes are commonly accompanied by atony, and should always be suspected in obscure cases. Ovarian cysts and uterine displacements in women, chronic prostatitis, and seminal vesiculitis in men are the lesions most usually found.

Whether atony of the stomach may result from irritative conditions of the intestine or colon is not as yet a proved fact, although probable from experimental and clinical observation. Symptoms of atony complicated by extreme degrees of constipation are often relieved by the expulsion of old fecal masses, by laxatives or enemas. Atony may apparently be induced by the continued use of irritating purgatives, and this probably is a frequent cause for the complaint in women. Cannon injected a few drops of croton oil into the cecum of cats and

the following day fed them with a bismuth potato meal. No potato entered the colon until six or seven hours had elapsed, and the food was still present in the stomach at the end of seven hours; whereas under ordinary conditions potato would be found in the colon within two or three hours and the stomach would be empty at the end of this time.

Symptoms.—Of all the symptoms of atony, flatulence is the one of which complaint most commonly is made. Flatulence may be either gastric, intestinal, or both. In the writer's cases gas in the stomach constituted the chief complaint in 77 per cent., intestinal distention alone in 51 per cent., while in but 12 per cent. distress from gas was slight or absent. Gastric and intestinal discomfort from flatulence occurred together in 40 per cent. of the cases.

Gastric Flatulence.—Gastric flatulence produces a sense of fullness, distention and discomfort. Actual pain from gaseous distention is so rarely observed that its occurrence should suggest a complicating pyloric spasm. The distress usually begins shortly after eating, continues for an hour or so, and gradually subsides. The degree of distress is dependent more upon the quantity of the meal than upon the character of the food that is eaten. The larger the meal, the greater is the distress. Water may cause as much discomfort as an equal weight of solid food. In mild cases the annoyance may be apparent only after the heartier meals, and may be slight or absent if smaller meals are taken, while in severer cases every meal, no matter how scanty its quantity, may be followed by the complaint. In the most advanced forms of atony, even small quantities of water may provoke the discomfort.

In mild degrees of atony there is a period of relief and comfort appearing two to four hours after eating and lasting until the next meal is taken, but in severer types of the disorder, distress is more continuous and the discomfort merges into that caused by the succeeding meal, so that at no time is the patient really comfortable. The gas may be freely raised and passed or the patient may be unable thus to find relief, the gas "stays fixed." It not infrequently happens that distention may persist during the greater part of the night, preventing sleep or waking the patient during the early morning hours. The degree of flatulence is almost invariably increased if the patient be overtired or more than ordinarily nervous. In many cases distress only occurs on the days of over strenuous living and excitement.

The tendency of the patient is regularly to attribute his discomfort to something that has disagreed, and he feels that this is so because with gaseous eructations he can "taste his food." The result is that he cuts off one article after the other until he is reduced to a starva-

tion diet, and by becoming weak and run down increases his atony to such an extent that he suffers as much or more with his insufficient diet as he did when he ate more liberally. Eructations of gas bearing the odor of food that has been eaten, means absolutely nothing unless the repetition of taste occurs hours after the ingestion of food at a time when the stomach should normally be empty. It is then an indication of tardy food expulsion rather than a test of whether the food is or is not properly digested. Inflation of the stomach by gas may induce nausea, or may embarrass the action of the heart and cause rapidity of the pulse, palpitation, and dyspnea.

Intestinal Distress.—Intestinal distress may be occasioned by the passage of gas from the stomach into the bowel or may be due to an associated atony of the intestinal wall. Abdominal distention usually appears three or four hours after the meal, occurring one or two hours later than the distress occasioned by the gas in the stomach, and is more continuous, so that it becomes an annoying feature of the disease during the night. Flatus may be easily passed, or the gas may simply “roll around.”

Heaviness and Sense of Weight.—Heaviness and sense of weight in the stomach were the cause for complaint in 42 per cent. of the writer's series. The patient feels that he has overeaten even though the meal be light and is inclined to sit still, as the upright position aggravates his discomfort. Lying down is equally distressing, as the stomach contents press up on the diaphragm and cause dyspnea and palpitation. The sense of heaviness is directly proportionate to the mechanical weight of what has been taken and is not influenced in the least by the character of the food.

Constipation.—The bowels are usually constipated. In the writer's series constipation was noted in 75 per cent., attacks of diarrhea alternating with constipation occurred in 15 per cent., while in 10 per cent. the action of the bowels was regular. Constipation if present is almost regularly due to a concomitant intestinal atony, often aggravated by an insufficient diet. The diarrheal attacks seem to be caused by recurring irritation from intestinal stasis or to the overuse of the cathartics to which the patient becomes addicted.

Nausea.—Nausea is present in about one-fifth of the cases. The nausea is rarely extreme, seldom if ever passing into the active stage of vomiting, neither does it seem to be closely dependent upon the taking of food, but more usually appears at any time irrespective of the meals and comes and goes throughout the day. It is not influenced, as a rule, by the character of the food. Occasionally nausea appears in sudden sharp attacks relieved entirely by eructations of wind from the stomach.

Appetite.—The *appetite* is rarely normal. The patient usually is fairly hungry at the meal, but after a few mouthfuls “feels stuffed,” so that he can eat no more with comfort. This early satiety is observed in nearly all the cases of atony. Aversion to food is more rare, as actual anorexia occurred in only 5 per cent. of the writer’s series. A feeling of being “all gone” in the region of the stomach may appear two or three hours after the meal. The patient “would gladly eat if he felt he could.” This occasioned complaint in 5 per cent. of the series.

Vomiting.—Vomiting is but rarely observed, except as a symptom of “sick headache,” although some of the neurasthenic patients may contract the habit of inducing vomiting to dislodge the gas. The vomited matters never contain traces of residual food of ancient date as in pyloric stenosis.

Heart-burn.—Heart-burn may be observed in a few instances, occurring in the writer’s series in but 4 per cent. Many patients bring up gas and with it a few particles of digesting food naturally and normally acid, and say that they have acidity and sour stomach. Their misapprehension on this point is obvious.

Headache.—Symptoms which we ordinarily ascribe to *auto-intoxication* are common with gastric atony, or more usually with a combination of gastric and intestinal atony.

Of these, headache constitutes the most common complaint and occurred in all of the writer’s cases. The most common form is the so-called “sick headache” or “bilious headache,” occurring periodically. Beginning usually over one or the other eye the pain becomes hemicranial in distribution, and is attended by nausea and vomiting. Scotomas and other visual symptoms are usually absent. In other instances, the headache is dull and occipital, usually more intense on awakening and passing off gradually as the day progresses.

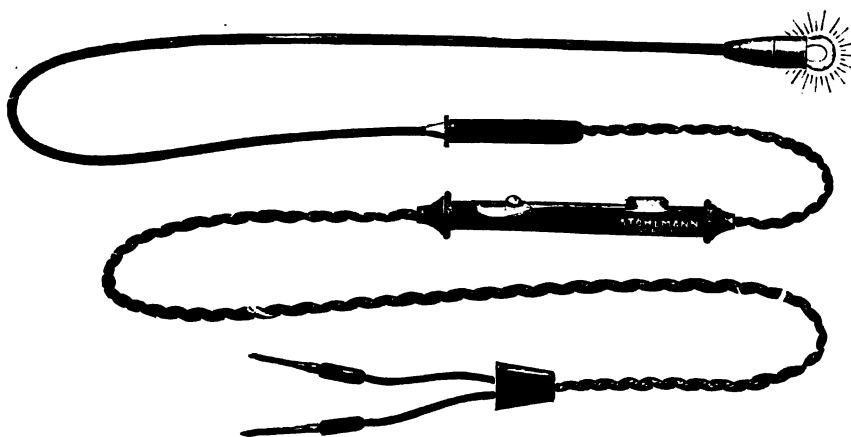
Dizziness is not uncommon, usually being characterized by its constancy rather than by its severity.

Many writers speak of morbid apprehensions and fears as characteristic of gastric atony. This has not been the writer’s experience, as in but 2 per cent. of his series was there any mention of any form of phobia. It would seem more probable that the morbid fears were due to the original psychasthenic state rather than to the atony that occurred in such a neurotic subject.

Diagnosis.—Physical Signs.—Atony may be present without any physical signs to reveal its presence, so that negative results on examination should not throw out the diagnosis. Repeated examinations are often necessary, as the condition may be more evident at some time than at others. The physical signs are usually more evident two or three hours after eating than at any other time.

1. Much valuable evidence may be afforded by the physical inspection of the patient. Stout, healthy looking individuals with broad costal angles are rarely subject to functional disorders of digestion and in such subjects atony is comparatively rare. Those who are delicately built, of highly sensitive nervous organizations, and who are thin and peaked, are more liable to the disorder, and especially is this the case with those whose costal angle is sharp and who present the other stigmas of the enteroptotic habitus. Gastropotosis is the strongest presumable proof of atony, and occurred in over one-third of the writer's cases of the ailment.

FIG. 56



Writer's gastrodiaphane.

2. An atonic stomach may not necessarily be a large stomach at all times, but it is a stomach that is abnormally distensible and lacking tone, tends to sag deeper and deeper as more food is put into it. Examination of the patient as he lies down may show that the greater curvature lies at or above the umbilicus, especially if the organ be comparatively empty. When he stands, however, and several glasses of water are taken, the lower curvature sags so as to reach 2 or more inches below the umbilicus. This is more clearly shown by x-ray examination than by the other means, for it is often very difficult to locate the lower curvature in the standing posture. Gastrodiaphany or the introduction of small electric lights into the stomach, so that they lie in the most dependent portion of the organ, would apparently be of service, as it would seem that the lower curvature of the stomach could be located by the point of maximum intensity of the transmitted illumination. The writer has been guilty of having devised a gastrodiaphane, but has

long since abandoned its use as being utterly untrustworthy for any purpose whatever,

Moderate inflation of the stomach is perhaps the most accurate means of locating its lower border, but it must be borne in mind that the stomach is so distensible that the more it is inflated the larger it becomes, so that entirely erroneous results may be obtained. Meinert made this error when he announced that almost all chlorotic girls had dilated stomachs. He simply overdilated distensible atonic stomachs that may not have been larger than normal if he had let them alone. Inflation in atony should be only to the extent of producing a change of note in the auscultatory percussion of the organ. Inflation greater than this is unnecessary, misleading, and unjustifiable.

3. Splashing sounds by percussion or palpation of the stomach are readily elicited in atony and may or may not be of diagnostic significance. Almost any normal stomach will produce percussion sounds when it is violently agitated at a time when it is full. In very thin subjects succussion sounds may be normally produced by deep palpation. With atony these so-called *deep* succussion sounds are of no diagnostic significance. Succussion sound produced by *light* palpation may be heard normally in women whose abdominal wall is greatly relaxed as the result of repeated child-bearing, but otherwise occurring in a patient whose abdominal wall offers an apparent normal resistance to palpation, succussion sounds that are audible by the slightest tapotement afford presumptive proof of atony. Succussion sounds in atony cannot be demonstrated when the stomach should be empty, but if after fasting they reappear after the ingestion of a half-glass of water, atony may be diagnosticated almost with certainty.

4. In atony visible peristalsis is never evident nor can gastric stiffening ever be demonstrated

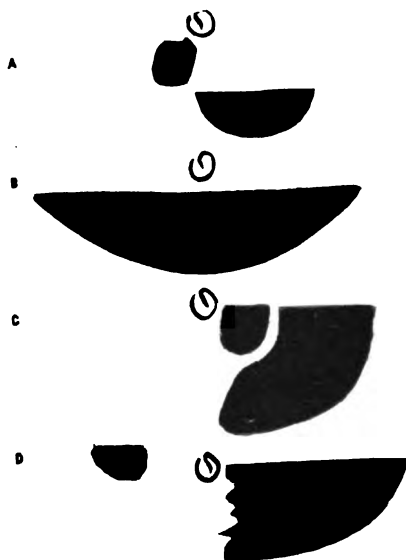
Radiographic Diagnosis.—The radiographic diagnosis of atony is based on the examination of two sets of plates, one taken six hours after the bismuth meal, the other directly after the stomach has been filled with the bismuth suspension after the six-hour plate has been taken.

Bismuth residue in the stomach six hours after the first bismuth meal may be due to pyloric stenosis, to ulcer of the lesser curvature, to cancer, to perigastric adhesions limiting free motility, or to atony. Large semilunar residues extending far to the right, may obviously be due to pyloric stenosis, but otherwise the differential diagnosis between these conditions cannot be made by the study of the six-hour plate above. The general outlines of the bismuth residue in the six-hour plates are shown diagrammatically by Holzknacht.

It must be remembered, however, that atony, to a clinical degree,

may exist with normal food expulsion—the lack of tonus not being accompanied by any corresponding lack of peristalsis. In such instances, the six-hour plate may show no bismuth residue whatever, so that a negative plate does not exclude atony.

FIG. 57



Residue after six hours. The position of the residue is shown in relation to the umbilicus: *A*, small residue due to atony, spasm of the pylorus, or slight stenosis; *B*, broad extensive residue, due to uncomplicated stenosis of the pylorus; noteworthy are the bowl-shape of the residue and its extension far to the right of the median line; *C*, "snail form," from shrinkage of the lesser curvature due to ulcer, residue far to the left with sharp bend of the greater curvature; *D*, residue displaced to the left, margin well defined and jagged, no displacement of pylorus, tumor of the pylorus. (Drawn from Handek, in Holzsknecht's article in *Archives in Röntgen Rays*, July, 1912, p. 69.)

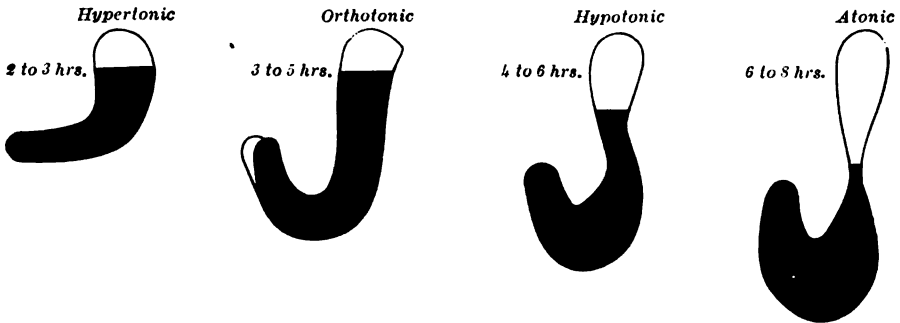
The second bismuth suspension meal given after the six-hour plate has been taken serves to show the outline of the filled stomach. The different appearances of the filled stomach are shown in the accompanying diagram from Holzsknecht.

"This classification," writes Holzsknecht, "according to the habitual tone of the organ gives us a method of testing the motility of the stomach far in advance of anything obtainable by the ordinary clinical methods. As may be seen in the diagrams the normal time for the complete evacuation of the stomach varies between two and eight hours. For types 3 and 4 a delay of six hours would be normal, whereas for type 1 it would indicate some obstruction in the pylorus. In type 4 even eight hours' delay would lead us to no suspicion of either spasmodic or

permanent contraction." In general Holzkecht is correct in the above conclusions.

It can hardly be said, however, that the hypotonic and atonic types can be included among those of the normal stomach. An atonic stomach is certainly not a normal organ. Neither must it be assumed in every instance of these atonic types that food evacuation is delayed, for as has been previously explained, lack of tonus, or atony, may or may not be associated with a corresponding reduction in peristalsis.

FIG. 58



Ordinary forms of the full stomach due to differences in tone, patient standing. The figures indicate in each case the time required for complete evacuation.

In atony the bismuth meal falls quickly to the most dependent portion of the stomach, lying transversely, and sagging the organ downward, leaving the upper portion empty except where the rugæ have retained the bismuth. The walls of the pars media are seen quite collapsed, appearing as a vertical cord. The lower portion of the stomach is well distended and displaced downward, lying well below the umbilicus in the upright position of the patient. The pylorus, however, will be at its normal point and the terminal portion of the greater curvature will sweep upward and to the right. The stomach bubble is usually of large size, although the bubble may be exceedingly small.

The shape of the stomach differs materially from the "snail form" of ulcer or cancer of the lesser curvature, and from the "undershot" appearance of the greater curvature as seen in pyloric stenosis.

The outline in atony is not irregular, nor indented, as with ulcer, cancer, or extensive adhesions. The differences between the appearance of the filling of the normal stomach and that with atony have been previously described.

Gastric Analysis.—The ordinary routine examination of the fasting stomach and test breakfast may afford but scanty proof of the existence

of atony, although it is serviceable in excluding other disorders. A third test has to be added which concerns itself in the length of time the food is retained in the stomach before it passes on its way. We have, therefore, to consider the fasting stomach, the test breakfast, and tests for motility.

FASTING STOMACH.—The fasting stomach in atony should invariably be empty. Demonstrable food remains or hypersecretion are never encountered in simple functional atony, and the atonic dilatation of the stomach described by older writers does not exist.

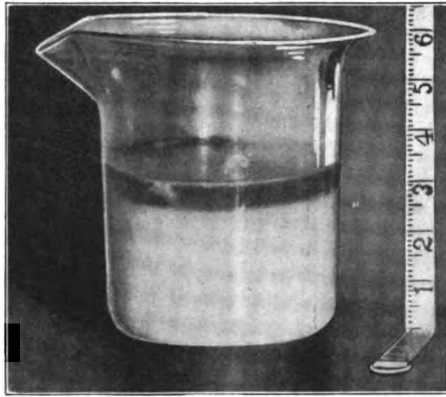
It is unfortunate that many writers speak of atony as muscular insufficiency of the first degree, and of pyloric stenosis as muscular insufficiency of the second degree, as if the former might merge into the second. Atony and pyloric stenosis are two distinct conditions, having no relationship whatever with each other. In pyloric stenosis the one invariable proof of its presence is the finding of food remains or hypersecretion fluid in the fasting state, whereas in atony the fasting stomach is empty. In 100 of the writer's cases in which examination of the fasting stomach was made, in 82 the stomach was absolutely empty, in 14 were found small quantities of fluid under 20 c.c., giving acid reaction but not containing microscopical or macroscopical food remains. Ten of these 14 patients were gastroparetic. In 96 per cent. of the cases, therefore, the fasting stomach was practically empty. In 4 patients only was there found fluid over 30 c.c., and in each of these 4 patients a chronic appendicitis could be demonstrated.

The *test breakfast* is often characterized by its abundance and by the increase in the amount of fluid which it contains. An excessive amount of test breakfast return is not, however, apparent in the majority of instances. In three-fourths of the writer's patients the amount of test breakfast removed was less than 65 c.c. It cannot be said, however, that the amount of test breakfast removed by aspiration represents the total amount of food contained in the stomach at the time of the test, for it is more difficult to aspirate the contents of an atonic stomach than if the organ be of normal tone. To determine the quantity of food remaining in the stomach after the withdrawal of the test breakfast, tests for motility may be employed, which will be shortly described. The test breakfast, on standing, usually separates into two layers, the upper fluid layer being equal in depth to that of the sedimentary layer in contrast to the normal test breakfast in which the supernatant fluid is rarely over half the depth of the sediment. The breadstuffs, as a rule, are well digested and homogeneous. It is said that in atony hyperacidity is the rule. This is not according to the writer's experience of 100 cases of atony:

Total acidity was	10 to 20 in	4 cases
Total acidity was	20 to 30 in	5 cases
Total acidity was	30 to 40 in	10 cases
Total acidity was	40 to 50 in	24 cases
Total acidity was	50 to 60 in	17 cases
Total acidity was	60 to 70 in	19 cases
Total acidity was	70 to 80 in	12 cases
Total acidity was	80 to 90 in	8 cases
Total acidity was	90 to 100 in	0 cases
Total acidity was	100 to 110 in	1 case

On analyzing this table it is seen that subacidity was present in 19 per cent., normal acidity in 60 per cent., hyperacidity in 21 per cent.

FIG. 59



Test breakfast in uncomplicated mild atony. The breadstuffs are well digested and unmixed with mucus. Acidity normal. Moderate hypersecretion.

TESTS FOR MOTILITY.—The older methods of testing the motility of the stomach by giving capsules of salol and iodipin which pass unchanged through the stomach, disintegrate in the duodenum, become absorbed, and appear in the urine or perspiration, are too inaccurate to require much consideration, and they have been totally abandoned. The only tests for motility are those by the *x*-ray and by the use of the tube.

Detection of bismuth meal within the stomach at different periods of time after it has been eaten is the only positive and accurate method of determining gastric motility, but it is unfortunately a method that cannot be used as a routine form of examination.

Riegel's test is based on the assumption that the normal stomach should be empty seven hours after a test dinner consisting of soup, beefsteak, and bread, but that in conditions of atony, remains of this meal are found in the stomach at the expiration of this time. Riegel's test is simple, and would be quite satisfactory were it not for the fact

that the examination of the patient exactly seven hours after a meal is often at a most inconvenient time. A simple modification of Riegel's test, employed by the writer, is as follows:

The patient is directed to take for breakfast at 7.30 A.M. a cup of coffee with milk and sugar, a chop or a small piece of steak, and a breakfast roll. Thereafter nothing is to be taken, not even water, until the examination at 1 P.M., five and a half hours after the meal. Under normal conditions the stomach is then empty or contains less than 20 c.c. of food remains. Quantities of residue greater than this indicate motor error.

Mathieu and Réymond have devised a test to calculate the amount of residue left in the stomach after the withdrawal of the ordinary test breakfast, by determining the acidity of the test breakfast itself, and the acidity of residual breadstuffs washed out by a definite quantity of water. The test breakfast is removed by aspiration at the proper time by the ordinary method, and the contents laid aside. A funnel is then attached to the tube and 200 centimeters of water are run into the stomach. The funnel is then lowered and raised, allowing the fluid to flow to and fro so that a thorough admixture of fluid and residual breadstuffs in the stomach takes place. The whole quantity is then aspirated and the total acidity of each specimen is determined. If "b" represents the quantity of the undiluted test breakfast and "a" its acidity, and if "q" represents the quantity of water introduced (in this instance being 200 c.c.), and a' the acidity of this diluted food residue, it is evident that the 2 acidities a and a' must be directly proportionate to the quantities, because the greater the quantity of water used for dilution the smaller is the total acidity of the diluted food. This ultimate formula is obtained:

$$x = \frac{a'q}{a - a'}$$

By adding this residual amount to the volume of the test breakfast originally withdrawn, the total amount of test meal in the stomach may be determined. For example, through the tube are withdrawn 60 c.c. of test breakfast. Having the acidity of 48, the acidity of the diluted contents is found to be 14. The formula is then expressed as follows:

$$x = \frac{14 \times 200}{48 - 14} = \frac{2800}{34} = 82$$

The total quantity therefore is 60 c.c., the undiluted contents plus 82 the diluted contents, or 142 c.c. of test breakfast remaining in the

stomach one hour after the meal has been taken. Under normal conditions, the total quantity of the test breakfast thus obtained should not exceed 200 c.c. Quantities exceeding this amount indicate motor insufficiency.

This test is not accurate when motor error is accompanied by hypersecretion, and should therefore not be employed in any case in which this complication is present. Being based on the estimations for acidity, it is naturally not applicable to those in which motor insufficiency coexists with an absolute of hydrochloric acid.

A very simple method of estimating the motility of the stomach has been suggested by Elsner and has been adopted by the writer as a routine test. The test breakfast is withdrawn in the usual manner, a Politizer bag containing 200 c.c. of water is then connected with the tube and the water injected. By allowing the fluid to be sucked back into the bulb and then reinjected several times, a thorough admixture of the water with the gastric contents takes place, after which the total contents are withdrawn and poured into a graduate glass. The original test breakfast is poured into another glass and both specimens allowed to settle, after which the amount of sediment in both specimens is determined. A total residue of 100 c.c. may be normal. Residues of 100 to 125 c.c. are on the borderline, quantities exceeding 125 c.c. indicate motor error.

By any of these tests for gastric motility, it may be demonstrated that the exit of food from atonic stomachs is tardy, although complete, if sufficient time be allowed to elapse. *In other cases, even though the subjective symptoms of the disorder are present, the food seems to leave the stomach within proper time limits, so that a negative result obtained by any of the tests for motility does not necessarily exclude gastric atony.*

Course.—The course of the disease varies from the expression of a slight and transient complaint to that of a chronic and harassing disorder.

1. Recurring atony may occur in individuals of nervous organization, especially in those who are burdened by the enteroptotic habit, at intervals throughout their entire life. These are the instances of delicate digestions, easily upset and slow of recovery. Such patients, generally neurasthenic, anemic, and undernourished, are always forced to live quietly and eat carefully to avoid trouble, and yet in spite of all their care they will suffer if they become tired or nervous.

2. Acute atony from transient causes is often overlooked. After anesthesia, atony is almost regularly present, but usually subsides within twenty-four hours. When postanesthetic vomiting persists after this time the existence of an enlarged atonic stomach can regularly be demonstrated. Atony may reappear on the third to fifth day after

the operation, dating from the time at which solid food was given. Gas, nausea, heaviness of the stomach after eating, and lack of appetite are the ordinary symptoms observed.

After sudden mental shocks, or excessive fatigue, the patient may feel too tired to eat. After the meal the food lies heavily upon the stomach, and occasions distress and flatulence. In these cases psychic disturbances of gastric secretion are often present, so that the food is improperly digested. Diarrhea may succeed such an attack. After several days the normal tonicity of the stomach may return, but in other cases the symptoms of atony exist for weeks after the apparent cause has disappeared. In acute illness a diminished and easily appeased appetite with gaseous distention of the stomach may indicate an existing atony. If too excessive quantities of liquid by mouth be allowed, prolonged or permanent atony may result.

3. When atony is once firmly established the symptoms are apt to continue without any cessation, less marked during the vacation months or at times of comparative ease, and more distressing during the times of greatest stress and responsibilities, but never interrupted by complete freedom for any length of time. Improvement may be expected from treatment, but the disorder is apt to recur whenever any extra mental or physical strain is thrown on the individual in the performance of his daily allotted task.

Treatment.—Atony is essentially a functional reaction of the stomach consequent upon the wear and tear of daily life, and the more we are impressed with this fundamental idea the better will be the results of our treatment. Every individual has his normal limitations, some more and some less, and our chief object should be to see to it that as far as possible the patient should live well within these limitations. Those with the enteroptotic habit and those with delicate sensitive organizations are especially unable to withstand shocks and trials of life. A careful personal inquiry should therefore be made as to daily routine, and every habit or circumstance that tends to diminish nervous or physical force should be corrected as far as may be possible. Physical or nervous expenditures should be compensated by adequate rest. No amount of medical treatment, however scientifically conducted, will prove beneficial if the patient be allowed to waste energy and strength and to use up nervous capital.

Dietetic Treatment.—The principle of the dietetic treatment is to conserve the muscular power of the stomach by not overloading the weakened organ by too much food at any one time, but to distribute the burden through the day by small and frequent meals.

The bulk of the weight of the food should not be increased by copious draughts of fluid taken with the meals. Restriction of liquids is an

essential part of the treatment. The patient may drink between meals if he be thirsty, but never more than half-glass at a time. The maximum amount of liquid at any one meal should not exceed one glass. Excessive drinking "to flush the system" should be absolutely interdicted.

The quantity of each meal depends largely upon the degree of the atony. In mild cases a meal smaller than the average, but with a restricted amount of liquids, may be taken three times a day at the conventional hours, and small meals allowed in the forenoon and afternoon. Eating at bedtime is generally inadvisable as it may increase the distention during the night and prevent sleep. Coffee may be allowed for breakfast; soup at luncheon or dinner should be prohibited. A sample diet for mild atony is as follows:

- 8 A.M. Cup of coffee, or cocoa, with cream and sugar, fine cereal.
- 11 A.M. Egg-shake, Russell's emulsion, or koumys.
- 1 P.M. Steak or chop, one vegetable, rice pudding, bread and butter.
- 4 P.M. Chicken sandwich and a glass of hot milk.
- 7 P.M. Fish or chicken, two green vegetables, tapioca pudding.

In more advanced atonies the bulk of the larger meals should be still further reduced, and if possible the size of the smaller meals should be increased. A sample diet may be thus given:

- 8 A.M. Cup coffee, or cocoa with cream and sugar, soft-boiled egg, bread and butter.
- 11 A.M. Baked custard.
- 1 P.M. Minced chicken on toast, corn-starch pudding.
- 4 P.M. Scraped beef sandwiches.
- 7 P.M. Small broiled chop, creamed spaghetti.
- 10 P.M. Cup of malted milk.

The quality of food makes very little difference if the bulk of the meal be reduced, as the digestive power of the gastric juice is normal or hyperpeptic in four-fifth of the cases.

Food that is tough, gristly, and indigestible should, however, in all cases be interdicted, as undigested lumps are rejected by the pylorus and remain abnormally long in the stomach. Thorough and leisurely mastication for the same reason must be enjoined, and the teeth put in good condition for the task. Hasty eating and the bolting of food are distinctly detrimental. If achylia should coexist, meats should be cut out from the diet and replaced by carbohydrates of equal caloric values as described in full under achylia.

If the gastric secretions are well preserved, meats may be given freely, although the writer usually interdicts the use of beef, and the heavier red meats, owing to the large amount of indigestible connective-tissue fiber which they contain, and replaces them by chicken, fish, lamb, or tender lean broiled or boiled ham. *Under no circumstances*

should the diet be so restricted that the patient loses weight. The tendency of atonic patients is to attribute their distress to what they eat and accordingly to reduce their diet. The more they starve the weaker they become and the more atonic. Patients should be encouraged to eat enough to maintain body weight, or preferably to gain in their nutrition, nor should the diet advised be so monotonous as to create a distaste for food. Individual tastes must be considered.

It has been recommended to place atonic cases on a purely milk diet, given 8 to 10 ounces every two hours so that 2 quarts are taken daily. The writer is absolutely and unalterably opposed to this line of treatment, as the coagula are firm and tough, owing to normal preservation of lab-ferment and are retained too long within the stomach, and moreover unless milk be given in at least double this quantity the total caloric value is quite insufficient to maintain weight and strength. Two quarts of milk represent less than 1500 calories, supposing the milk to be of extra richness, whereas at least 2500 calories are required to meet the demands of body expenditure.

When the diet is conducted on these lines, there is usually a marked improvement in the distress and distention. In other cases no improvement whatever can be noted so long as the patient is up and around. A week or so of absolute physical rest in bed is then indicated, and usually is attended by satisfactory results.

Medical Treatment.—Medical treatment is of less importance than the dietetic or physical. Theoretically nux vomica or strychnine are indicated to increase the motor power of the stomach, and although often disappointing in its results, apparent benefit follows the use of this drug in many cases. The tincture or extract of nux vomica or similar preparations of physostigma may be employed, and these preparations seem to be somewhat more efficacious than either strychnine or eserine in isolated form.

The following prescription may be employed:

R—Tincture physostigma	3v
Elixir calisaya	ad 3iv
M. Sig.—Teaspoonful in a wineglass of water after meals three times a day.	
R—Tinct. nux vomica	3v
Sod. glycerophosphate	3v
Elixir diazyme (Fairchild)	3iv
Aquæ	ad 3viiij
M. Sig.—Tablespoonful in water after eating, three times a day.	

Should anacidity coexist, diluted hydrochloric acid or oxyntin may be employed. When gastric secretion is maintained the degree of acidity is rarely excessive, nor is a continuous hypersecretion apt to occur,

so that it is rarely necessary to give alkalies. Bicarbonate of soda or magnesia may, however, be advised should the patient complain of heart-burn or pyrosis.

Anemia should be combated by preparations of iron, though large doses should not ordinarily be advised. Constipation should be controlled by diet and massage rather than by drugs if possible. When laxatives are necessary, the mildest forms of medication should alone be used and in the smallest possible doses. *Every medicinal laxative tends to perpetuate atony.* Salines should be administered cautiously. Enemas though having their disadvantages seem to the writer less objectionable than overmedication by mouth. The quantity injected should not exceed 2 pints, and the enema given lying down. Enemas given in the sitting posture are not to be advised.

The writer has endeavored to stimulate intestinal peristalsis by the use of peristaltic hormones.

Fairchild has prepared for the writer some preparations of the hormones: secretion (Starling and Bayliss), which he has used for some time, and more recently the peristaltic hormone of Zuelzer. He has used these two hormones in combination with the bile salts (Fairchild) with good results. This combination has been prepared in capsules, each containing bile salts $\frac{1}{2}$ gr., secretion 1 gr., hormone peristaltic 10 gr.

The peristaltic hormone has also been used in injections prepared in ampoules, each containing 5 c.c. The sterile fluid (in physiological salt solution) is taken up into the sterilized ampoule and immediately sealed.

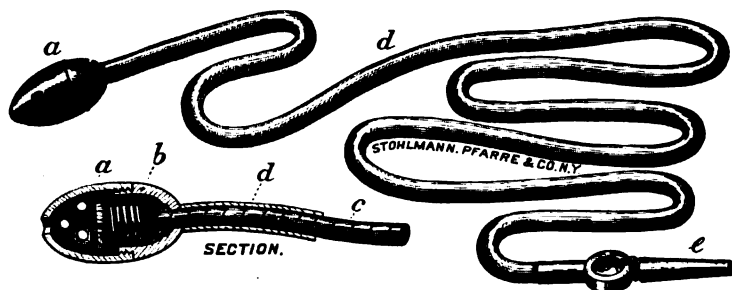
In the present state of our knowledge of the chemistry of the hormones and the fact of the ready susceptibility of these principles to change, these special preparations have been prepared in small quantities only from time to time and sent directly from the laboratory to the pharmacist as required.

Physical Treatment.—1. Rest is to be insisted on in every case as an antidote for overactivity. Rest should regularly succeed exercise, especially before eating, so that the patient is refreshed at the time of the meal. It may be necessary for the patient to be in bed for at least two hours after the mid-day meal before any improvement from the treatment is apparent. Exercise directly after eating should always be prohibited. In severe degrees of atony in run-down individuals, a rest cure becomes a matter of necessity and should be conducted along the lines recommended under the treatment of gastropnoia.

Intragastric faradism was formerly employed more frequently than at the present time. The writer's intragastric electrode consists of a perforated hard-rubber capsule containing a metallic tip to which is

attached a spiral of fine piano wire covered with rubber tubing, so as to be both small in caliber and extremely flexible. The electrode is easily introduced and creates after one or two introductions little or no discomfort. A large moist pad is then placed over the epigastrium and a slowly interrupted faradic current passed, of sufficient intensity to produce visible contractions of the abdominal wall. The action is increased by the sipping of water from time to time. The applications should never be made within two hours after eating, and the duration of the seance should not exceed fifteen minutes. The writer had a long experience in intragastric faradism, at first employing the treatment with an enthusiasm, which has steadily diminished, so that at the present time he has practically abandoned its use.

FIG. 60



Writer's intragastric electrode.

In gastric atony lavage is contraindicated, as the introduction of water in bulk tends to sag and overdistend the weakened organ. In the majority of cases of atony, food is well digested and not mixed with sufficient gastric mucus to call for the mechanical cleansing. If demonstrable mucous gastritis coexists, lavage may be employed, but the majority of water induced at any one time should be very small, not over 8 ounces, and care should be taken that an equal quantity is reclaimed. Mineral waters have no place in therapy of atony; they are distinctly and absolutely contraindicated.

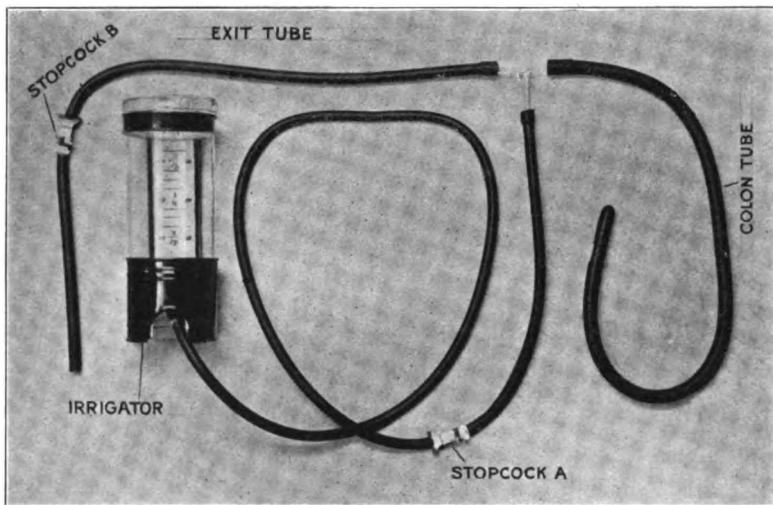
Massage is of considerable service if skilfully done. Deep and forcible massage with the ends of the fingers should be prohibited and the physician should be careful that the massage is not too violent.

Hydrotherapy is a valuable addition to the treatment. A simple expedient is for the patient on rising in the morning to lie for a moment or two in hot water and then take a cold shower or to pour cold water from a pitcher down the back while ten is slowly counted. A strenuous course of hydrotherapy in institutions devoted to this form of treat-

ment is not generally advisable, as the patients are apt to react badly after the first stimulating effect has passed.

The symptoms of intestinal auto-intoxication are often improved by intestinal irrigations. Irrigations are often given by the use of two rectal tubes, the inflow being through one, the outflow through the other. While water is easily introduced into the bowel, it is not so easy to get rid of it unless the exit tube be of large caliber so that it does not become blocked. One such tube is quite enough to pass into the bowel at one time; hence, the writer's objection to the double tube. A single large tube should be used with a to-and-fro current. The size of tube often employed is quite too small and becomes easily blocked, so that the water introduced remains in the bowel, much to the patient's discomfort. The writer's apparatus for intestinal irrigation is as follows:

FIG. 61



"Author's irrigation outfit. By closing stop-cock *B* and opening stop-cock *A* the fluid enters the colon. After a sufficient quantity has been introduced by closing stop-cock *A* and opening stop-cock *B* the fluid flows out into a large collecting jar."

A rectal tube of the requisite size is attached to a glass T-tube, one of the other arms of which is attached to an irrigating jar of at least one liter capacity, the other to a soft-rubber tube about 4 feet in length. Pinch cocks are placed on the inflow and outflow tubes. The rectal tube is then inserted about 6 to 8 inches, and the apparatus held in position on the side of the bed by long pins. The outflow cock is closed and about one pint of water allowed slowly to fill the bowel. The inflow cock is then closed and the outflow cock opened so that the water is

allowed to run out, and the process repeated. Six to eight quarts of water are used at each treatment and an estimation made of the difference between the amount that is injected and the amount that is returned so that the difference representing the retained fluid does not exceed one pint.

FIG. 62



Author's colon tube. Actual size. Notice large caliber and ample size of the openings for good drainage.

FIG. 63



Author's modification of the Kemp tube.

An irrigation so given demands the assistance of a nurse or attendant and therefore is not always practicable.

For purpose of home use without the help of an attendant the writer has devised a modification of Kemp's tube, by increasing the vertical dimensions of the instrument, by enlarging the eye, and by carrying the inflow tube along the upper portion of the instrument so that it does not block the centre of the eyes, as does Kemp's instrument.

CHAPTER XI

ACUTE DILATATION OF THE STOMACH

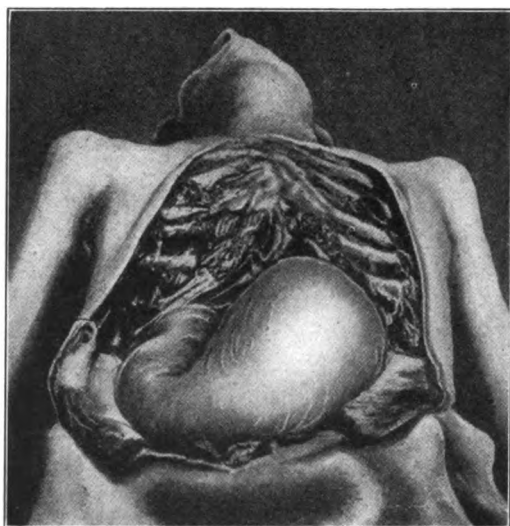
Synonyms.—Arteriomesenteric ileus, acute gastrectasis, duodeno-jejunal obstruction, postoperative gastric dilatation, gastrojejunal obstruction, duodenal ileus, mesenteric ileus.

Acute dilatation of the stomach was formerly regarded as somewhat of a rarity, Laffer, in 1908, having found but 217 cases recorded in literature. The reported cases up to this time were chiefly those with a fatal outcome, the mortality being 63.5 per cent. of the cases in which the symptoms were distinctive and obvious. During the last few years, however, numerous cases have been reported of undoubted acute dilatation, in which the symptoms while characteristic were less severe and eventuated in recovery. It is even believed at the present time that many instances of postoperative vomiting are examples of this disorder. When these facts therefore are taken into consideration we are led to the belief that acute dilatation is not at all an uncommon event.

Pathology.—The most striking phenomenon observed at the autopsy is the enormous dilatation of the stomach so that it may occupy nearly the entire abdomen and may even extend to the pelvis. The dilated organ is usually bent into two unequal portions, one comprising the greater part of the stomach, extending almost perpendicularly downward, and a smaller portion consisting of the pyloric and extending upward and to the right at an acute angle, giving the dilated viscus a V-shape, which has been likened to a fat arm flexed. The surface of the stomach may be congested and covered with dilated bloodvessels or may be pale in appearance. In a little over half the cases the duodenum is dilated in whole or in part. In the majority of instances the duodenal dilatation stops at the point where the duodenum is compressed between the root of the mesentery and the superior mesenteric artery in front, and the aorta and vertebral column behind. In other cases the dilatation extends past this point to the lower duodenum or even the upper portion of the jejunum. The intestine below is almost invariably empty and collapsed, there being but one case (Mahomet's) in which the intestines were dilated throughout their course. The pylorus is regularly patent and no organic stenosis is found in the lumen of the duodenum.

At the spot where the root of the mesentery and its contained superior mesenteric artery and veins cross the duodenum there is normally a moderate degree of compression exerted sufficient to hold back the bile and pancreatic juice for a certain time after meals. When the stomach begins to empty itself one and a half to three hours after eating, the entrance of chyme into the duodenum causes sufficiently strong contractions of the duodenal wall to overcome the resistance at the compression point and to force its contents into the jejunum. Under abnormal conditions this mesenteric compression may become so extreme that the muscular forces above the point are powerless to overcome the obstruction, and dilatation of the stomach and the duodenum

FIG. 64



Acute primary dilatation of the stomach associated with pneumonia. (From Thomson.)

proximal to the compression point ensues. The most common cause for an increased mesenteric compression of the duodenum is traction on the root of the mesentery by a descent of the intestines into the pelvis. This mesenteric constriction can be experimentally produced by traction on the root of the mesentery by a weight of 500 gm., equivalent to that of the small intestine.

Mechanism of Acute Dilatation of the Stomach.—The mechanism of acute gastric dilatation is an interesting subject which has been provocative of much discussion and argument, but about which at present time we know comparatively little. Two conflicting opinions are expressed:

1. That the dilatation is due to an occlusion of the duodenum resulting from its compression by the traction of the root of the mesentery, or by the pressure of the superincumbent and dilated stomach on those parts of the duodenum which lie in contact with the front and left side of the spinal column. In other and rarer cases, the dilatation may be produced mechanically by duodenal kinks.

2. That the dilatation is the result of paresis of the gastric wall either peripheral and analogous to the meteorism of the intestine that occurs with typhoid fever, or central and due to the diminished motor impulses conveyed to the stomach through the vagus nerve.

The various causes suggested may be tabulated as follows:

1. Mechanical causes.

- (a) Arteriomesenteric constriction (favored by lordosis).
- (b) Duodenal kinks.
- (c) Mechanical weight of stomach on duodenum (favored by counterpressure as in plaster jackets).
- (d) Cardia closure by folds or intragastric pressure.

2. Paralytic causes.

(a) Central:

- 1. Section of vagi.
- 2. Involvement of vagus in pneumonic exudate.
- 3. Blows or head injuries.

(b) Peripheral:

- 1. Operations near stomach with traumatism or slight sepsis.
- 2. Post-anesthetic.
- 3. Overdistention by excessive eating and drinking.
- 4. Toxemic.

Mechanical Theory.—(a) The mechanism of mesenteric constriction has been admirably described by Conner,¹ and by him is regarded as the most important cause for acute dilatation.

For the entrance of the intestine into the pelvis, according to Conner, three things are necessary: a dorsal decubitus, an intestine nearly or quite empty of gas and feces, and a mesentery of sufficient length to enable the intestines to slip into the pelvis. There is no doubt that a mesenteric occlusion of the duodenum may actually occur, for in 19 out of 38 cases in which the duodenum was dilated, actual compression was found. In this connection a case reported by Baumler is important, for at autopsy he found a band of bright red color 2 cm. broad on that part of the duodenum which ran under the root of the mesentery and the duodenal mucosa corresponding to this band showed super-

¹ Amer. Jour. Med. Sci., March, 1907, p. 345.

ficial pressure necrosis. Compression of the duodenum by the root of the mesentery is favored by a forward curve of the vertebral column as in lordosis.

Although it is regarded by many that the duodenal compression thus induced is the primary cause for gastric dilatation, there is some doubt as to whether such an occlusion may not be the *result* of the dilatation rather than the cause. In enteroptosis we have, as is well known, sagging downward of the small intestines and traction of the root of the mesentery, but in spite of this we have no dilatation of the stomach and no backward filling of the stomach by biliary secretions. In 250 of the writer's cases of gastropptosis in which examination of the fasting stomach was made, in not a single instance was there any evidence of a backward regurgitation into the stomach comparable with the fluid vomited by those who suffer from acute gastric dilatation.

It is claimed that fasting and purgation enforced before an operation allow the intestine to become empty and collapsed so that it sinks readily into the pelvis and pulls on the root of the mesentery. This may be so, but there are many other instances of a similar collapsed condition of the intestine without mesenteric constriction, such as after the vomiting and purging of acute gastro-enteritis, or the collapsed intestine of starvation, or after any severe or continued diarrhea. Moreover, acute dilatation is not infrequent with pneumonia, in which disease abdominal tympanites rather than intestinal collapse is more usual.

Other mechanical causes besides mesenteric constriction have been found to explain the dilatation in other cases, and were noted in 8 of 38 cases collected by Conner in which the dilatation ceases at the lower duodenum. There may be found sharp kinks at the duodenojejunal junction. Such a cause was found in Petit's case and was relieved by lifting up the jejunum and stitching it to the transverse mesocolon with complete recovery of his patient. In other cases occasional kinking at the junction of the first and second portion of the duodenum has been noted. Angulation at this point is often experimentally produced by the forcible dilatation of the stomach of a cadaver with air, but rarely resists a water pressure of over 20 c.c.

Box and Wallace¹ have reported five cases in which at autopsy the dilated heavy stomach lay upon the duodenum and compressed it. The gastric dilatation was evidently maintained by the pressure of the stomach on the duodenum, since on raising the former gas immediately rushed into the jejunum. Box and Wallace called attention to the fact that the compression must of necessity be greater before the abdominal

¹ Lancet, July 22, 1911.

cavity is open, owing to counter-pressure exercised by the abdominal muscles, and for this reason experiments on the cadaver with the stomach exposed only reproduce in a very imperfect manner the conditions present during life.

Kelling considers that a valve-like closure of the cardia by folds of mucous membrane, which is favored by the oblique insertion of the esophagus into the stomach, may result whenever the stomach is experimentally inflated, and he suggests that this cardia closure in dilatation of the stomach may render it impossible for the stomach to empty itself of its contents. There can be no doubt that in some of the recorded cases air and fluid were contained in a dilated stomach at high pressure. In one of Laffer's cases, when the tube entered the stomach a great quantity of gas whistled out, followed by a jet of black fluid that squirted three feet. This could hardly have been the case had the cardia been normally patent.

Mechanical overloading of the stomach may be followed by its paralytic overdistention. Grundzach's¹ patient collapsed from this condition after eating 30 hard-boiled eggs and drinking a considerable quantity of wine. Minor attacks may follow overeating by gluttonous children.

Paralytic Theory.—*Paresis of Central Origin.*—As the motor impulses to the stomach are conveyed through the vagus nerve, any condition which interferes with this function of the nerve may be followed by gastric dilatation. Paralytic dilatation of the stomach in dogs has followed section of the vagi at different levels. Several instances are reported of gastric dilatation following injury to the head, supposedly from paresis of the motor function of the vagus. The frequency of dilatation occurring during the course of lobar pneumonia is explained by some, by involvement of the vagus trunk, by exudation into the posterior mediastinum.

Paresis of Muscular Wall.—Paresis of the muscular wall of the stomach may result from prolonged operative procedure in the neighborhood of that organ and may therefore be considered an evidence of local traumatism.

Postanesthetic Paresis.—Postanesthetic paresis is supposed to be an occasional cause. We must consider in this connection the animal experiments made by Kelling and Braun. These experimenters inflated the stomach of dogs upon whom gastrostomy had been done and found that whenever a certain degree of distention was reached vomiting reflex was excited and the stomach was emptied by eructation and by vomiting. When, however, the animal was narcotized the vomiting

¹ Wien. med. Presse, 1897, No. 43, p. 1350.

reflex was abolished, and the stomach could be distended to the point of bursting without the least escape of air or stomach contents through the esophagus.

After recovery from the anesthetic there is usually a hyperexcitability of the vomiting centres. After a variable time the vomiting centres become again normal, but occasionally it is thought conditions of exhaustion or even of abolition of function may occur which may conduce to acute dilatation. When an anesthetic and an operation are associated it is difficult to say which is the more responsible for the event.

Toxic Paresis.—Toxic paresis may be autogenous or exogenous.

Autogenous toxemia producing muscular paresis of the stomach wall may result from auto-intoxication by poison generated within the alimentary canal. While this cause has been adduced by some, the writer has not been able to satisfy himself that it has been an operative cause in any of the reported cases of which he has knowledge.

Exogenous toxemia is becoming recognized as probably the most important factor in producing acute gastric dilatation. According to this theory, the infective toxins of pneumonia or typhoid fever, or of the other forms of septic infection act as paralyzers of the gastric musculature and allow of atonic dilatation. Very interesting in this connection is a recent communication by Rutz,¹ in which is described a number of cases of pneumonia in which pneumococci were found in the stools associated with a considerable degree of abdominal distention. According to this writer the pneumococci or their toxins act locally upon the wall of the alimentary tract to produce a moderate degree of paresis with resulting distention.

The number of cases reported of acute dilatation of the stomach complicating pneumonia is increasing every year and constitutes one of the commonest types of the disorder. There is no reason in these cases for supposing that the intestines shrink and drop into the pelvis, thus causing traction of the root of the mesentery. In pneumonia there is more usually a moderate degree of abdominal distention, which should hold the intestines in place. It is probable, therefore, that the dilatation is due to the direct effect of the toxemia and that any duodenal occlusion is a secondary affair.

Laffer's 4 cases were all septic, one postpartum case with sloughing of the abdominal wall following injection of salt adrenalin solution, the second followed abscess of the antrum with pyemia and staphylococci in the blood and left knee, the third occurred with gangrenous appendix, while the fourth complicated pyelitis with cystitis and pyelitis.

¹ New York Med. Jour., July 20, 1912, p. 113.

Of the cases reported by Box and Wallace in which the weight of the stomach on the duodenum was apparently the cause for the dilatation, 3 were decidedly septic. The writer is impressed on reading the reported cases with the remarkable frequency with which acute dilatation occurs in the course of various forms of infectious diseases or conditions of sepsis. In abdominal operations the septic element may not be sufficient to call forth evident general symptoms of such, but nevertheless slight degrees of peritonitis may exist, scarcely more than an injection of the serosa, but quite sufficient to cause a definite muscular paresis. In a case reported by Halsted¹ dilatation of the stomach and first portion of the duodenum followed operation for gallstones. Accurately corresponding with the distended portion of the bowel was a slight peritonitis with just enough exudate to cause adhesions between the duodenum and gall-bladder.

Summary.—In review of what has been said concerning the mechanism of acute dilatation and the various causes that have been adduced to explain the condition, it is evident that no one single cause can be applied to all the cases. The following deductions can be formulated.

1. In rare instances mechanical dilatation of the stomach may be induced by excessive eating or drinking either by causing a paralytic overdistention or by mechanical pressure of the overloaded stomach upon the duodenum.

2. Mechanical pressure of the stomach on the duodenum is favored by counter-pressure on the abdominal wall, explaining the relative frequency with which the accident has occurred after the encasement of the body by a plaster jacket in orthopedic cases.

3. Arteriomesenteric constriction by traction of the root of the mesentery from downward displacement of the intestines may occur in rare instances as a primary cause for acute dilatation, although when the stomach is dilated the enlargement of the organ tends to push the intestines downward and to create a mesenteric pull that is quite enough to keep up an obstruction after it has been once started. This view implies, therefore, that in the very great majority of instances mesenteric constriction is a secondary factor in the production of an acute dilatation.

4. The same may be said of mechanical obstruction of the duodenum by the pressure of an overloaded stomach on the duodenum; in the majority of instances the dilatation is the first event and the mechanical pressure of the stomach is entirely a secondary affair.

5. Diminished motor innervation by paresis of the vagus is a plausible reason to explain the occurrence of acute dilatation following head injuries.

¹ Johns Hopkins Hosp. Bull., January, 1900, p. 16.

6. Lowered tone of the vomiting centres after anesthesia as a cause, may be considered doubtful.

7. Dilatation of the stomach may be increased after the process has once started, by closure of the cardia either by valve-like folds of mucous membrane or by lateral intragastric pressure on an oblique insertion of the esophagus.

8. Probably the most potent cause for acute dilatation is a paralytic relaxation of the gastric wall due to the effect of various toxins, in infectious diseases and in septic conditions. A primary dilatation so induced may lead to a certain degree of mesenteric constriction which is often a marked contributory factor.

9. Local peritonitis of the gastric serosa with muscular relaxation may occur after abdominal operations, not sufficiently intense to cause septic or frank inflammatory symptoms but quite sufficient to induce an appreciable degree of dilatation which may be further increased should any of the secondary factors for dilatation be brought into play.

Etiology.—The various exciting causes for acute dilatation may be inferred from what has been already written concerning the modes of origin of the complaint.

Of 217 cases compiled by Laffer, 97 (44.7 per cent.) followed an operation which in 60 instances was a laparotomy. The occurrence was most frequent after operations on the biliary system (15 cases). Next in frequency came operations of the kidney (11 cases) and of the appendix (5 cases). In but 4 cases did it follow operations in the stomach itself.

In 11 instances dilatation followed various operations on the extremities.

The time of onset after operation is difficult to determine, as the actual onset may be masked by postanesthetic vomiting. In less than one-half the cases the symptoms were apparent on the first day of the operation, although the third and fourth day furnished the larger number.

Traumatism has been thought to be a cause in 17 cases in Laffer's series, but of these only in 5 instances was the force applied to the abdomen.

The occurrence of dilatation in orthopedic patients after the application of a plaster jacket occurred in 5 instances.

A certain degree of atony regularly follows anesthesia, the lower curvature of the stomach extending below the line of the umbilicus. Such an atonic condition should subside in twenty-four hours, but occasionally it may continue and produce characteristic symptoms of acute dilatation, especially on the third or fourth day, when the patient begins to take solid food.

Excessive eating or drinking was an alleged cause in 20 of Laffer's

series. The dilatation has occurred in three instances after a seidlitz powder had been given by the physician to distend the stomach for the purposes of examination.

An increasing number of cases are being reported in which dilatation complicates the progress of some infectious disease or septic process. Pneumonia seems to head the list, localized or disseminated tuberculosis comes next in frequency. By far the greater majority of cases of acute dilatation give such an antecedent history of disease. As earlier and milder forms of the disease are now recognized, this complication is not as infrequent as we have been led to suppose.

Symptoms.—The characteristic symptoms are the repeated vomiting of copious bile-stained fluid and a demonstrable inflation of the stomach.

Vomiting is usually the first indication of the disease and is present in 90 per cent. of the cases. Very characteristic of dilatation in post-operative cases is the vomiting of bilious fluid at a time when post-anesthetic vomiting should have ceased. The few patients who do not vomit suffer the most distress, as the organ becomes more rapidly overdistended and the true condition is more readily overlooked. The vomited matters consist of a dark green or black flocculent fluid, either odorless or foul. In rarer instances, especially in the cases complicating pneumonia, the odor may be distinctly fecal. The quantity raised by any one attack of emesis may not be large, but the vomiting is so incessant and uncontrollable that the total quantity of fluid lost becomes quite rapidly excessive, the patient vomiting "basinfuls" during the day or night. The act of vomiting is rarely accompanied by much straining, but the fluid wells up in large gulps without much effort. In rarer instances it may be projectile. The vomiting may be continuous or may disappear for hours, or even for several days. The cessation of vomiting is not to be regarded as a favorable sign unless an improvement is also noted in the patient's general condition and unless physical examination shows a marked diminution in inflation. Unless these favorable signs are present cessation of vomiting usually indicates a diminished power of contraction of the stomach requisite for the vomiting act, and implies an increasing accumulation of fluid within the stomach.

Examination of the vomited matters show almost invariably the presence of bile and of pancreatic ferments. Hydrochloric acid may be present and may even be in excessive amounts, but, as a rule, the reactions for free hydrochloric acid are absent, possibly owing to neutralization by the alkaline fluids of the duodenum. Lactic acid is often present. Streptococci and other organisms are often found in the vomited matters.

Pain is usually severe and is proportionate to the degree of distention.

Abdominal tenderness may or may not be present. Occasionally acute dilatation of the stomach, even though it be extreme, runs an entirely painless course.

Collapse with the Hippocratic facies usually appears early in the severe cases, and the patient presents a rapidly developing picture of severe illness. The tongue is dry, the pulse rapid and feeble, the face assumes a pinched expression, the temperature is apt to be subnormal.

Thirst is excessive and cannot be assuaged. Dyspnea and embarrassed action of the heart are occasioned by the upward pressure of the dilated stomach.

In less severe cases the general condition may remain good for a considerable period of time and the pulse may be surprisingly regular in frequency and of good strength. This condition of well-being is often quite misleading, so that the physician may minimize the significance of the bilious vomiting and epigastric distention, and underestimate the danger until it is too late, for unless the condition be recognized and properly treated the collapse symptoms are apt to appear sometimes earlier, sometimes later, and too often are the precursors of a fatal issue.

In mild cases the symptoms while characteristic may be much less marked. Many postoperative cases complain on the day after operation of abdominal pain and distention, and of the repeated vomiting of bilious greenish fluid. The stomach on examination is found to be moderately enlarged. These symptoms last a few days, often regarded merely as instances of prolonged postanesthetic vomiting, and then gradually subside. The postural treatment is usually followed in these mild cases by complete disappearance of symptoms within a few hours.

Minor degrees of dilatation may occur during the course of lobar pneumonia, characterized only by abdominal distention and bilious vomiting. These are the early symptoms, and if neglected, severe abdominal distention may occur rapidly, associated with painless effortless vomiting and collapse.

Physical Signs.—Epigastric distention is usually obvious. The inflation of the stomach is most marked at first in the left hypochondrium corresponding to the vertical arm of the distended organ, later the distention spreads to the epigastrium and downward so that the whole abdomen is visibly protuberant, occasionally in the postoperative cases, tearing out the stitches and allowing the wound to gape.

Succussion sounds and splashes are usually distinctly audible when the stomach is sharply palpated or percussed, and an apparent fluctuation may even be obtained by bimanual palpation.

An increase in peristalsis has been visible in a small number of the patients that were said to be suffering from acute dilatation of the

stomach. It is a question whether in acute dilatation increased peristalsis in any form can be demonstrated unless the acute dilatation be engrafted upon a more chronic obstruction of the pyloric canal or duodenum.

Rigidity of the abdominal muscles does not occur as in peritonitis.

Diagnosis.—The diagnosis is most readily made by the passage of a stomach-tube. The escape of air in large quantities, the outflow of a fluid having the characteristic previously described, and the flattening of the epigastrium after the stomach has been thus emptied of its gaseous and fluid contents, are signs of the greatest diagnostic significance, especially in the cases which have not undergone a previous operation.

The diagnosis of postoperative dilatation from postoperative ileus high up in the alimentary tract due to adhesions or kinks is often impossible, except by the course of the disease and the improvement which may follow postural treatment and lavage in the case of gastric dilatation. If the fluid withdrawn from the stomach by the tube clearly indicate its origin in the small intestine the diagnosis would naturally incline toward intestinal obstruction. The more pronounced the fecal odor of the vomitus the greater the chances of actual obstruction. Intestinal obstruction high up in the alimentary tract may present symptoms so closely resembling those of acute gastric dilatation that a differential diagnosis is well-nigh impossible.

In certain instances chronic obstruction suddenly becomes acute and complete. In these cases the patient usually gives an antecedent history of crampy pains, recurring attacks of abdominal distention and vomiting and other signs which may be indicative of neoplasm or ulcer.

General intestinal distention not due to obstruction or peritonitis, general peritonitis, pancreatic cysts, uremia, are conditions from which gastric dilatation should be diagnosticated. In most of these conditions the passage of a tube will not afford relief as it does in acute gastric dilatation, while in uremia there is no distention.

Prognosis.—Prognosis is always grave. In the earlier reported cases mortality exceeded 70 per cent. Of Laffan's series 62.5 per cent. died, but more recently under improved methods of treatment the mortality has decreased to 53 per cent. (Payer). Of 11 cases of acute dilatation complicating pneumonia, reported by Fussell, 5 recovered, 6 died. In the series from which these statistics were compiled the symptoms of the disease were severe so that the diagnosis was evident. Milder cases which are now frequently recognized were not included. It is probable that as the conditions become better recognized and the therapeutic indications more widely known that the disease will cease to be as formidable as it is now regarded.

Treatment.—The treatment of acute dilatation seems to be now based on correct principles, and if the disease is recognized sufficiently early, medical treatment is often followed by extremely brilliant results. It is to be expected, moreover, that by an early and rigid enforcement of these medical principles of treatment the mortality of the disease will be very considerably lessened. The treatment is to be conducted on three principles.

1. To interdict all food and nourishment.

2. To wash the stomach.

3. To cause the patient to lie in such a position that the mechanical weight of the stomach and the direct effect of mesenteric traction are overcome.

1. All fluids and nourishment of any kind by mouth should be absolutely interdicted. There is no use in adding to the fluid contents of the stomach when the presence of the fluid contents is doing harm, and, moreover, there is very little chance indeed that any fluid or food given by mouth can pass into the intestines and become absorbed.

2. The stomach-tube should be passed at frequent intervals and the stomach emptied either by aspiration or lavage. The tube should be passed far enough to reach the level of the fluid in the stomach, and it is well to make a mental calculation that the tube should be passed far enough to reach nearly to the pelvis. An attempt to wash or empty the stomach should be made even though the patient be apparently moribund. Collapse is no contraindication, nor should one hesitate even in the presence of extensive and presumably fatal pneumonia. It may be urged that there is no apparent reason for emptying the stomach in cases of repeated and copious vomiting, on the ground that the stomach is by emesis sufficiently emptying itself. This argument is entirely erroneous, because the passage of a tube even after emesis will show that a great deal of fluid still remains in the stomach, the patient only vomiting the surplus. Lavage should be frequently performed, in severe cases even every two and three hours, in less severe cases two and three times a day. One is guided as to the frequency of lavage by the quantity of fluid obtained at every procedure and upon the apparent relief afforded, as shown by the increased comfort evinced by the patient and by an improvement in his general condition. After lavage the epigastrium usually flattens itself, and an indication for the repetition of the procedure is the return of epigastric distention. Continuous drainage has been recommended by the passage of a soft, rather small tube through the nostril into the stomach and retained in place by tape and adhesive plaster. The fluid can be started by compression of a bulb inserted in the rubber tube of the apparatus, and siphonage so started can be successfully continued for a number of hours. The

writer sees no advantage in continuous drainage, but prefers the ordinary method of lavage frequently repeated.

3. The postural treatment is of the utmost importance, and is designed to relieve mesenteric traction and the weight of the superincumbent overloaded stomach upon the duodenum. The dorsal decubitus must be positively prohibited, and the patient should be forced to lie on the right side or on the abdomen. Improvement often follows the right-sided or abdominal posture within a few hours and the symptoms often recur in an equally short period if the patient reassume the dorsal decubitus. The knee-chest position is theoretically the best posture for the relief of mechanical conditions, but is usually impossible after laparotomies or during the course of severe pneumonia. The lateral or ventral decubitus seems to be more effective when the foot of the bed is raised.

Medicinal Treatment.—Drugs are of very little service. Theoretically eserine or physostigma should be of service. Eserine, gr. $\frac{1}{10}$, may be given hypodermically every two or three hours and may be recommended as a routine measure, although not very much is to be expected from its use.

Apomorphine to empty the stomach by emesis has been recommended, but this therapeutic indication is better met by lavage than by the use of so depressing a drug as this.

There seems to be no physiological indication for atropine, although it has been suggested as a drug that may be of service. Colon irrigations are often of the greatest service in supplying fluid to the system and thus improving the general condition of the patient and mitigating to some extent the torments of his thirst as well as stimulating gastrointestinal peristalsis.

Surgical treatment of acute dilatation is now considered inadvisable. Gastro-enterostomy has been performed on the ground that mesenteric obstruction produces a duodenal stenosis which can only be treated by surgical means, and that, moreover, gastro-enterostomy is a drainage operation. The general consensus of opinion in the present day is decidedly against such an operation. The only excuse for surgery is in those postoperative cases in which a differential diagnosis between acute dilatation and acute obstruction by reason of adhesions and kinks is impossible to be made. Under these circumstances unless the conditions improve by lavage and the postural treatment it may be justifiable to explore.

CHAPTER XII

PYLORIC SPASM AND PYLORIC STENOSIS

Pyloric Spasm.—Spasmodic closure of the pylorus as a pure neurosis seldom if ever occurs. Contraction of the pyloric sphincter, or more properly speaking, reflex inhibition of pyloric relaxation, is regularly secondary to or symptomatic of an irritation in its neighborhood, either on its gastric or its duodenal side, or it may occur as a protective spasm in irritative lesions of the midgut or its derivatives. Gastric and reflex causes are thus recognized.

Etiology.—Gastric Causes.—Temporary pylorospasm may occur from irritation of the prepyloric portion of the stomach by coarse undigested or improper food. The pyloric sphincter is possessed normally of a selective control of the food which it allows to pass. As the result of gross dietetic error, the pylorus may contract to prevent improperly digested or irritating masses of food from passing into the duodenum. This form of pylorospasm is common in the experience of nearly everyone. After the offending meal has been eaten the patient will complain of pains and cramps in the stomach, followed by nausea, heart-burn, and the vomiting of food residue admixed with acid fluid, often in greater quantities apparently than the amount of food recently ingested. The attack is terminated by the emptying of the stomach, and no further trouble is experienced unless dietetic errors be repeated.

Pylorospasm with Ulcer.—Pylorospasm occurs with ulcer, acute or chronic, in the neighborhood of the orifice, either gastric or duodenal. With acute ulcer or erosion the condition is analogous to the spasm of the anal sphincter occasioned by fissure at that orifice. In acute ulcer the symptom of pylorospasm may be first evidence of disease, while in other cases the spasm does not occur until the ulcer symptoms are well established, and may even appear during the ulcer cure. *Pylorospasm from acute ulcer* is characterized by acute hypersecretion. The patient complains of a lump, oppression, or distress appearing after meals which lasts more or less continuously during the attack. Relief by eating is slight and temporary. Comfort is often afforded for a time by alkalies, although very large doses are required. Nausea and vomiting commonly occur, the vomited matters being liquid in character, exceedingly acid, and containing food remains that have remained in the stomach for an abnormal period. The vomitus is often of a

brownish color, due to altered blood. In many cases the most marked distress occurs during the early period of the night. Complete relief comes only after vomiting or the emptying of the stomach through a tube, but the distress usually reappears after the lapse of several hours.

The symptoms of pyloric spasm and acute hypersecretion are given in greater detail under the heading of Ulcer.

In chronic ulcer at or near the pylorus, gastric or duodenal, pylorospasm may occur from time to time, causing exacerbations in the symptoms of the disease. A constant and definite amount of pyloric obstruction may be usually demonstrated in the course of chronic ulcer from inflammatory or cicatricial thickening of the pylorus, but exacerbations of unusual severity are usually due to recurring pylorospasm which still further diminishes the lumen of the pyloric outlet. The symptoms so induced are largely due to increase in the hypersecretion. Patients complain of an increase in pain, distress and heart-burn, and vomiting of food and acid fluid, the fluid vomiting continuing even though all nourishment by mouth be discontinued.

The symptoms may continue for several days and then subside, or the added obstruction and the vomiting caused by it may precipitate a fatal issue.

Reflex Pylorospasm.—Reflex pylorospasm may occur from any irritative lesion in the course of the embryological midgut or its derivative, but is of special frequency with lesions of the gall-bladder and the appendix. Lesion of the gall-bladder may produce either a gastric atony or a pyloric spasm. The symptoms of atony have been elsewhere described. Pylorospasm is recognized by pain and by the phenomena that attend hypersecretion, pain, distress, acidity, pyrosis, and the finding of acid fluid in the fasting stomach.

With *cholecystitis* and *cholelithiasis* a spasm of the entire stomach may occur during the acme of pain, and undoubtedly intensifies the agony. The possibility of such an occurrence is proved by the x-ray examination of the stomach of a patient during an attack of gallstones described by Schlesinger.¹

The diagnosis of cases presenting this symptom complex suggests ulcer in the neighborhood of the pylorus, and our treatment may be entirely misdirected if we fail to elicit the characteristic physical signs of cholecystitis. Comparative rigidity of the head of the right rectus and stiffening of the costal arch are suggestive of a gall-bladder origin of the disorder, and our suspicion is strengthened if local tenderness be elicited over the gall-bladder, or if a palpable enlargement be found in this situation. Unfortunately these signs, indicative of chole-

¹ Berlin. klin. Woch., June 24, 1912.

cystitis, are not always present, or they may be present at some times and not at others, so that repeated examinations may be necessary before the diagnosis can be established. For further details the reader is referred to gall-bladder dyspepsia, page 576.

Pylorospasm induced by irritative lesions in the appendix is a common form of gastric complaint, and has been recognized as a clinical entity only in the last few years.

To Moynihan, the Mayos, and Paterson we owe a debt of gratitude for their contributions on this subject. The symptom-complex which they described appears in literature of the present day under the title of "Appendix Dyspepsia." It has been gradually recognized that in many intractable cases of indigestion where a lesion of the stomach, duodenum, or gall-bladder was supposed to exist, no structural alteration could be found at the time of operation, but on further examination an appendix obviously diseased is found to exist, and its removal is followed by a complete relief of all former indigestion.

Moynihan¹ noted that in many of these cases spasm of the pylorus was evident at operation, and in 1904 described the condition as follows: "On several occasions during the last few years I have watched the stomach intently for several minutes, and have seen the onset, and acme, and the gradual relaxation of a spasmodic muscular contraction in its walls. Quite gradually the stomach narrows, and the wall becomes thicker and almost white in color; when taken between the fingers the contracted area feels like a solid tumor. The spasm may be so marked as to prevent a finger being invaginated through the segment affected."

When this condition is seen it may be predicted that a lesion will be found in the appendix. The pylorus, therefore, acts as a guard to the bowel distal to it, and prevents the passage into the duodenum of food that might still further irritate the bowel condition. For further details of the symptoms of appendicular dyspepsia see page 568.

Treatment.—In acute pyloric spasm due to dietetic error the chief therapeutic indication is to empty the stomach of its contents, either by emesis or by washing of the stomach. Hot applications over the epigastrium are usually grateful to the patient. After such an attack the stomach should be given a rest by withholding food for some hours.

When pylorospasm occurs in the course of acute ulcer, we have not only to control the muscular spasm but to remove or neutralize the excessive amount of acid fluid which is the result of hypersecretion. If the patient be used to the tube the stomach may be drained of its

¹ British Med. Jour., 1904, i, 414.

irritating contents by ordinary aspiration, or by washing out the stomach with an alkaline solution. Unless, however, experience has shown that the passage of the tube is easy in a given case, the tube should not be passed, but alkalies should be given in sufficient doses to neutralize the acid fluid and to diminish the gastric distress. No food or drink should be allowed by mouth. Hot applications over the epigastrium are of the greatest service in reducing the spasm and afford relief to the patient. Atropine, hypodermically administered, should be given in small repeated doses until the point of physiological tolerance is reached. Doses of gr. $\frac{1}{40}$ may thus be given every three hours until its effects are evident.

In chronic ulcer a complicating pylorospasm should be controlled by placing the patient during the exacerbation of his ailment upon a liquid or semiliquid diet, such as that given during the second week of the von Leube ulcer treatment. Alkalies should be administered in sufficient doses to relieve the acidity and the distress. The atropine treatment may be employed. In some of these cases the oil treatment may be followed by considerable improvement. A tablespoonful of olive oil or liquid paraffin, or a 3 per cent. solution of anesthesin in oil, may be given a quarter of an hour before eating. In other cases a wineglassful of oil may be taken at bedtime. The patient should preferably be kept in bed until the severity of the attack be passed, and hot applications over the epigastrium should be continuously applied unless the patient has recently suffered from an attack of hematemesis.

For pylorospasm dependent upon gall-bladder affections, the above lines of treatment may be carried out with the addition of Carlsbad water, of urotropin, gr. $7\frac{1}{2}$ in a glass of hot water, half an hour before eating, or hot draughts containing 10 gr. of sodium salicylate before meals.

For pylorospasm of appendicular origin operation is indicated.

No operation done for the relief of intractable dyspepsia symptoms referred to the upper abdomen is complete without examination of the appendix and its removal if obvious signs of disease be present.

PYLORIC STENOSIS

Mechanism.—The mechanism of gastric digestion is so admirably poised that the muscular power of the stomach is sufficient to force the digested food at the proper time into the duodenum. Under normal conditions, peristaltic waves run continuously over the stomach, forcing the contents toward the outlet as long as the organ contains any food. The discharge from the stomach through the pylorus is not, however,

steady, but intermittent, as it takes place only when the appearance of acid chyme in the pyloric antrum causes the sphincter to relax. When the pyloric sphincter is unable to dilate to its full extent, by reason of spasm or cicatricial contraction, or of infiltration of its wall by inflammatory or neoplastic tissue, the resistance to the onward passage of chyme is necessarily increased. An additional resistance is encountered whenever actual constriction or closure of the outlet occurs by the narrowing of the orifice often to such an extent as to barely permit the passage of a slate-pencil. If the contraction be slight and not too rapidly progressive, the resistance to which it gives rise may be overcome, either by forcible contraction of the stomach wall, or by hypertrophy of the muscular tissue. The increased resistance in front is compensated by an increased force from behind, so that the required amount of work is accomplished. This compensatory balance is analogous to the hypertrophy of the left ventricle of the heart, with stenosis at the aortic orifice. Should, however, the resistance in front be too great to be overcome by an increase of gastric peristalsis, or by sufficient hypertrophy of the muscular tissue, the motor function of the stomach becomes impaired and the organ can no longer empty itself in the interval between the meals as it should.

The one characteristic and pathognomonic sign of pyloric stenosis is, therefore, the finding of food remains in the stomach at a time when that organ should normally be empty. This is the one and only infallible sign and symptom of pyloric stenosis.

Two resulting conditions must be considered.

1. As a result of food retention and the stimulation of the secretory nerve apparatus by the peptone bodies of digestion, as has been so admirably demonstrated by Pawlow and his school, there regularly occurs a secretion of gastric juice, continuous in the sense that it is poured out in excess of the quantity required, as long as there is any food lying in the stomach. This hypersecretion adds greatly to the bulk of the gastric contents, produces a symptom complex of its own, and gives to the vomited matters and test breakfast a fluid consistence that is quite characteristic.

2. As long as the stomach can empty itself of the greater part of its contents within a reasonable time, there is room within the stomach for the small accumulation of residual food and surplus gastric juice, together with the nourishment and fluids taken at the regular meals. When, however, the residual contents become excessive, the mechanical effects of excessive bulk and weight begin to be evident, the stomach loses its power of resilience, its walls become stretched and flabby, and there is a dilatation of the organ commensurate with the extra burden which it has to carry.

The word "dilatation" in this sense requires a word of explanation. The term is usually applied to all large flabby stomachs irrespective of their actual motor power. There are large, baggy stomachs which expel their contents within proper time limits, and there are normal-sized stomachs which are unable to empty themselves as they should. The size of the stomach has nothing whatever to do with its motor power. There are numerous examples of pyloric stenosis with food stagnation without any change in the size of the stomach from the normal, and in many cases of cancer of the pylorus, especially in those of rapid development, dilatation may be absent throughout the entire course of the disease.

Etiology.—Pyloric stenosis may be divided into two general groups—benign pyloric stenosis, and the malignant form, of which the type is cancer. The various causes for the two forms may be thus tabulated:

1. Benign pyloric stenosis.

(a) External pressure or traction.

1. Adhesions.
2. Pressure of external tumors.

(b) Contraction of the orifice.

1. Cicatrix from ulcer.
2. Thickening of pyloric wall by connective tissue, tubercle tissue, syphilitic tissue.
3. Benign tumor formation in the pyloric canal, fibromyoma.
4. Blocking of the lumen by pediculated tumors.

2. Malignant pyloric stenosis.

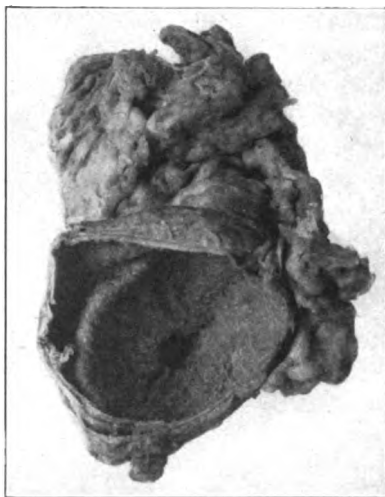
Tumors of malignant character, infiltrating the pylorus or encroaching on the lumen.

Benign Pyloric Stenosis.—Perigastric adhesions may bind the pylorus to the neighboring parts and interfere mechanically with its patency. Adhesions between the pylorus and the gall-bladder or the under-surface of the liver are the most important. The pylorus is frequently drawn sharply upward to the point of attachment, and acutely angulated. Tumors in the neighborhood of the pylorus and enlarged glands in the hilum of the liver may often by direct pressure interfere with the proper propulsion of chyme. The most frequent examples of extragastric tumors causing pyloric stenosis by reason of their pressure are enlarged gall-bladders, heavy with stones, which lie like sandbags upon the pylorus.

Contraction of the orifice is most commonly due to the cicatrization of a healed or healing ulcer at the pylorus. Not only is the lumen actually contracted in size, but the infiltration of the wall of the pyloric canal by dense scar tissue renders relaxation impossible. To this

organic stenosis there is often added from time to time a spasm of the pyloric sphincter, further diminishing the lumen of the orifice. Acute inflammatory tumefaction may also appear from acute exacerbations of the ulcerative process, and intensify the difficulty, while adhesions binding the pylorus to neighboring parts interfere with its mobility, and cause kinks and bends which further increase the mechanical obstruction. *It is important to remember that in the healing of ulcer any or all of these stenosing factors may be present in combination*, organic contraction, cicatricial infiltration, and perigastric adhesions being irremediable by medical treatment, while spasm of the sphincter and inflammatory swelling may subside under appropriate treatment. Herein lies the hope and the despair of the purely medical treatment of the disease.

FIG. 65



Benign pyloric stenosis following ulcer, from the duodenal side. (From Bloodgood's collection of specimens in the Surgical Pathological Laboratory of the Johns Hopkins Hospital.)

Infiltration of the walls of the pyloric portion often occurs with circumscribed cirrhosis of the stomach, with tuberculous deposits, and with syphilitic lesions of the stomach either in the form of ulcer with infiltrated base or with gumma or with diffuse syphilitic infiltration. These have been described under their respective headings.

Benign tumors having their origin in this portion of the stomach may often occlude the orifice, and are elsewhere described in detail.

It has occasionally happened that polypoid tumors of the stomach with a sufficient length of pedicle may engage in the pyloric opening, forming a ball valve which blocks the orifice completely. Obstruction of the pylorus by foreign bodies or masses of hard vegetable fibers

or of hair and by concretions due to medicines, such as bismuth, administered in large quantities and over long periods of time, has been known to occur, but the obstruction in these instances is usually intermittent, rarely continuous.

It is well to remember that changes in the duodenum may occur similar to changes in the pylorus itself and may be productive of gastric dilatation. This is especially true of ulcers and cicatrizations above the ampulla of Vater. Duodenal stenosis below the ampulla may be recognized by the constant presence of bile and pancreatic juice in the fasting stomach, a phenomenon quite different from the analysis of stomach contents obtained in cases of obstruction above the point at which the bile and pancreatic ducts enter the duodenum.

FIG. 66



Adenocarcinoma of pyloric end of stomach, showing extreme degree of pyloric stenosis. Patient alive and well three years after exsection. (From Bloodgood's collection in the Surgical Laboratory of the Johns Hopkins Hospital.)

Malignant Pyloric Stenosis.—Malignant stenosis practically means cancer, as the other form of malignancy, sarcoma, is relatively rare. Interference with the motility of the stomach by cancer may be

due either by implication of the pylorus itself by the neoplasm or by extensive infiltration and adhesions of the gastric wall cutting off normal peristalsis over a large area.

FIG. 67



Cross-section showing carcinomatous stenosis of the pylorus. *P*, very much stenosed pyloric canal; *C*, carcinoma infiltrating submucosa and muscularis; *S*, greatly thickened submucosa with carcinoma; *M*, muscularis. (From the Pathological Museum, Columbia University, New York.)

Symptoms.—The symptoms of pyloric stenosis may be divided into those:

1. Due to increased gastric peristalsis.
2. To the hypersecretion which is the result of food retention.
3. To food stagnation within the stomach.
4. To the diminished amount of chyme that enters the bowel for absorption.

I. Increased Peristalsis.—Pain and distress are the inevitable results of an increased peristalsis and occur with greatest intensity when the peristalsis is most active. In mild cases the patient will complain of a sense of fulness and discomfort, usually occurring two or three hours after meals, so that the patient may be reluctant to eat because of the resulting distress. The discomfort may be mitigated by soda, but is not completely relieved, as is the case with ulcer. Eating gives but slight and temporary relief, and eventually is followed by an increase in the distress. Temporary but incomplete relief may also follow the eructation of gas. In the severer cases the distress amounts to an actual pain, either sharp and cutting or cramp-like in character. These

pains continue during the period during which the stomach is endeavoring to force its contents through the constricted outlet. If the stenosis be but moderate the stomach may succeed in emptying itself more or less completely, so that succeeding the period of pain, comes gradually a period of relief, but later as the stomach succeeds less frequently and less completely in emptying itself there may be no interval during which pains may not appear, unless the stagnant contents be removed by lavage or by vomiting.

II. Hypersecretion.—Hypersecretion occasions heart-burn and eructations of acid fluid and is described in full detail under hypersecretion, page 510. These symptoms are not in themselves significant of pyloric stenosis unless they appear when the stomach should be empty. There is no other disease in which the time at which the examination is made is so important. Temporary relief is afforded by alkalies taken in sufficient doses to neutralize the large amount of acid fluid in the stomach, and it is suspicious of obstruction at the pylorus if the patient should wake at three or four o'clock in the morning and take large doses of soda for his relief. The distress from heart-burn and pyrosis often leads the patient to induce vomiting, as he finds that often his physical comfort can only be brought about by emptying the stomach in this way.

III. Food Stagnation.—The retention of food-remains in the stomach past the normal time limit sooner or later progresses to a stage in which the stomach is more or less filled with the food taken at one meal before the regular time for the patient to eat again. The food stasis, therefore, becomes collective and accumulative. The signs of fulness and satiety induced by the partially filled stomach at the time of the meal abolishes the normal appetite and may even create distaste or a loathing for food. This is especially so with cancer of the pylorus.

Occasionally with benign forms of obstruction due to cicatricial ulcer there may be a craving for food to neutralize the excessive hypersecretion, but this instinctive desire to eat is usually satisfied early in the meal.

Sooner or later vomiting is apt to occur. It may be that before spontaneous vomiting occurs the patient will attempt to obtain relief by inducing emesis, or if scientifically inclined, by washing the stomach. Many neurasthenics contract the habit of emptying their stomachs upon the occasion of any slight distress, but this is usually done during the period of active digestion. Those with pyloric stenosis are more apt to induce emesis a longer period than this after eating, in the majority of cases during the night or the early morning. As the residual food increases, spontaneous vomiting finally occurs and presents four characteristics that are quite diagnostic of pyloric stenosis.

1. The vomited matters consist of large quantities of food remains and liquid, far greater in volume than the amount of food and liquids introduced into the stomach for a long period of time preceding the emesis. The patient often wonders "where it all comes from."

2. As the vomiting represents the last desperate attempt of the overloaded stomach to empty itself, it is apt to occur some hours after the meal, usually between midnight and early morning. If it can be demonstrated that the patient repeatedly vomits food in the morning before breakfast, not having eaten since the meal on the previous evening, pyloric obstruction may be safely diagnosed.

3. In the vomited matters may be found particles of food that have been eaten many hours or even several days previous, that have been lying in the stomach for this length of time. Food may be recognized as having been eaten by the patient days or even weeks previously.

4. The vomited matters are quite liquid in character and gush out without much apparent effort.

The appearance and composition of the vomited matters are the same as of the contents of the stomach in the fasting state, and will be described in detail under the heading of Gastric Analysis.

It is important to remember that in certain cases vomiting does not occur, and therefore that the absence of this symptom does not necessarily throw out pyloric stenosis.

IV. Starvation.—The symptoms produced by the scanty amount of digested food that can pass into the bowel are largely those of lack of nourishment and dryness of tissue.

Loss of Weight.—Loss of weight is proportionate to the degree of stenosis and to the frequency and profuseness of the vomiting, the two most important factors which diminish the amount of nourishment entering the bowel for absorption. In advanced cases the patient may be literally skin and bones, weighing often not more than 60 to 70 pounds. These are, however, extreme and neglected cases, fortunately not as common in these days as they were some years ago when the diagnosis had not been perfected and when surgery had not reached its present development. Acetonemia is not uncommon in these extreme cases.

Constipation.—The bowels are usually obstinately constipated, the fecal masses being hard and scybalous. Attacks of diarrhea may occur from time to time and are due to the irritation of the bowel from the passage into them of decomposed stomach contents. In the liquid movements sarcinæ may be occasionally found. These attacks of colicky diarrhea may appear long before there are any gastric symptoms of disease, and are not, as a rule, correctly diagnosed, although examination of the fasting stomach will often reveal the true nature of

the ailment. This is another example of the importance of estimating the function and secretions of the stomach in all cases of obscure intestinal trouble.

Decrease of Urine.—The urine is diminished in proportion to the lessened amount of intestinal absorption and the profuseness of the vomiting. Usually the amount voided is in the neighborhood of 1000 c.c., but in extreme cases the quantity may be reduced to 400 c.c. The urine shows the characteristics of concentration, is rich in phosphates and poor in chlorides. Acetone and diacetic acid may be present in advanced cases of intestinal starvation. The blood is concentrated and is therefore relatively rich in the number of red blood corpuscles and the percentage of hemoglobin. Thirst may be excessive, even though the stomach be filled to the cardia, and is not assuaged by drinking.

Dryness of Tissues.—The dryness of tissues may result in numbness in the fingers and toes, muscular cramps, especially in the calves of the legs, which may be the precursors of gastric tetany.

The constitutional symptoms of benign stenosis are practically those of slow starvation. In malignant disease of the pylorus we have in addition the symptom complex that attends malignancy wherever situated. These symptoms have been described in detail under the discussion of cancer and need not be here repeated. It is sufficient to state in this connection that general symptoms due to malignancy, if present, are of the utmost diagnostic value in deciding whether in a given case we are dealing with a benign or a malignant form of pyloric obstruction.

Unfortunately cancer in its operable stage may not give the least evidences of its malignant character, so that in many if not in the majority of instances the diagnosis can only be made on indefinite but suspicious signs, symptoms, and analyses.

Gastric Tetany.—In a very few cases of advanced stenosis gastric tetany develops. The symptoms of this justly dreaded complication begin with pricking and numbness of the hands followed by carpopedal spasms. The finger tips may be brought together in the form of a cone, so that the shape of the hand is that assumed by obstetricians in their examinations. The attacks may last for several hours and may return with increasing frequency. At any time these slight symptoms may develop into classical convulsions. Temporary disturbances of intellect, lapses of memory, and disturbances of vision have been described.

Trousseau's phenomenon in tetany consists in precipitating a fresh attack by forcibly compressing the nerves or bloodvessels of the extremities. Tonic spasm of the affected extremity usually begins within one or two minutes and ceases when the pressure is withdrawn.

Erb's phenomenon is characterized by the increased irritability of the peripheral nerves to both faradic and galvanic currents, with the exception of the facial nerve. A minimum strength of current produces vigorous muscular contractions.

Chvostek's phenomenon is marked by an excessive muscular irritability to mechanical stimulation. Percussion with the finger or hammer over the nerve trunks is followed by lightening-like contractions of the muscles innervated by the irritated nerves. This phenomenon is most clearly evidenced by the contractions produced by tapping over the facial nerve.

Many are the theories advanced to explain the occurrence of gastric tetany. Many of them, such as Kussmaul's theory of dehydration of tissue, and Germain Sée's theory of reflex action from stimulation of the sensory fibers of the stomach, have been long since abandoned. Nothing is definitely known about its causation, although the theory of auto-intoxication from the products of the fermenting stagnating gastric contents appears to be the most reasonable one as yet advanced.

Intermittent or Latent Stenosis.—The course of the disease may either be steadily progressive, especially in the malignant cases, or it may be interrupted by periods of apparent improvement. Although structural stenosis may remain unchanged, varying conditions of tumefaction and spasm of the pyloric sphincter may temporarily increase the mechanical difficulty so that the symptoms become acutely aggravated. If the organic element of the stenosis, such as the degree to which it is rendered impervious by cicatricial contraction, be relatively slight, so that by increased muscular force the obstruction may be overcome, such a condition of compensation may not be attended by stasis, nor by any other characteristic sign or symptom, although a careful history will usually elicit the fact that there is often a sense of fulness and discomfort following the meals and occasionally even more severe epigastric pains. In this stage of comparative latency the condition may extend over months. These are almost regularly cases of benign stenosis, although occasionally even malignant pyloric stenosis may run such a comparatively quiescent course.

From time to time, either from inflammatory swelling or from pyloric spasm, symptoms of a more definite character occur. The patient will complain of attacks of epigastric pain, often lasting for several days. The attacks may follow gross errors in diet or may follow periods of intense mental or physical strain. The pain varies from one that is dull and aching in character to sharp colicky paroxysms. During the acme of pain epigastric tenderness is not infrequently extreme. The patient finds relief either in inducing vomiting or washing the stomach, the contents so evacuated being copious, watery, usually intensely

acid, and containing food remains that have been retained in the stomach long past the normal time limits, or seeks mitigation of the distress by abstinence from all food for twenty-four to thirty-six hours, thus allowing the overloaded stomach to empty itself gradually in the natural way without further adding to its burden. During the attack the outline of the stomach may be distinctly visible, gastric stiffening, visible peristalsis, and succussion sounds long after the last meal may be obtained on examination, and the passage of the tube withdraws fluid and food remains characteristic of pyloric obstruction.

After such an attack of sudden decompensation is over, conditions of apparently fair digestion are resumed, and the patient eats a reasonable quantity of food with impunity. Examinations of the fasting and digesting stomach may show no apparent departures from the normal. These cases are often spoken of as latent or intermittent pyloric stenosis, or the *stenose méconne du pylore* of French writers (Oettinger).

An interesting example of this intermittent course is as follows:

E. N., aged seventy years, was well until ten years ago, when he complained for about a year of pain in his stomach two or three hours after eating, lasting until he vomited, or ate again. He was then free from all distress until fifteen months ago, when for a week he had more or less constant epigastric pain and copious vomiting of a clear exceedingly acid fluid which brought temporary relief. After this attack he experienced no further trouble until three months ago, when acute symptoms again appeared. He would feel comparatively well during the morning and would eat his accustomed breakfast and lunch. In the middle of the afternoon severe gastric pains would appear and would grow so intense as night approached so that he was unable to eat any dinner. Toward midnight, when the pains were well-nigh unendurable, he would vomit with but little effort 2 or 3 pints or more of a scalding acid fluid containing in suspension the greater part of what he had eaten in the earlier part of the day. After the stomach had been thus emptied he would feel perfectly comfortable and would sleep soundly throughout the remainder of the night. After he passed through such an experience for several successive nights his symptoms would disappear for possibly two or three weeks before the occurrence of another attack.

In the interval between attacks, examination of the fasting stomach showed the presence of 55 c.c. clear fluid with only a few microscopical food remains. Total acidity 58, free hydrochloric acid 42. The abstracted test breakfast consisted of 95 c.c. of well-digested bread-stuffs without any trace of food previously taken. Total acidity 78, free hydrochloric acid, 40.

The patient was not seen for three months after this, his first examination, because he felt perfectly well and considered his indigestion at

an end, gaining 11 pounds during this time and eating everything he wished without discomfort. Suddenly and without warning the nocturnal vomiting reappeared. On the third day of his distress he was seized with intense abdominal pain and the attendant symptoms of perforation were quite evident. Operation was performed six hours after the accident and a perforation was found in the base of an old ulcer exactly at the pyloric juncture. The pyloric ring was thickened by cicatricial tissue and would barely admit the passage through it of a lead-pencil. The perforation was closed, gastrojejunostomy performed, and the patient made an uninterrupted recovery.

In a few instances pyloric stenosis may exist even to an extreme degree without producing any symptoms until decompensation suddenly occurs. The symptoms of pyloric obstruction appear suddenly and are often so severe as to suggest complete blocking at the outlet. The symptoms once started in full force do not pass away after a few days as in the former class of intermitting stenosis, but grow daily more marked, so that the patient will die within a few days of the onset unless relieved by a timely operation.

Such an example of a latent course with the sudden accession of alarming symptoms may be briefly cited.

K. J., aged forty-three years, was well until two weeks ago, when she began to complain of a moderate heart-burn two hours after eating, relieved temporarily by food, but completely alleviated by soda. Three days ago without apparent cause she began to vomit everything that she ate, the vomitus being acid, copious, and watery.

Fasting stomach contained 30 c.c. of liquid and food remains, the liquid being three times the depth of the sediment. Total acidity 44, free hydrochloric acid 16. Sarcinæ were present in great profusion. No lactic acid nor any blood.

Test breakfast 250 c.c., fairly well digested, three-quarters of the settled contents being fluid. Total acidity 130.

A tumor the size of a horse-chestnut was palpable in the pyloric area.

An immediate operation was performed by the late Dr. W. T. Bull, and a thickened mass was found invading the pyloric region and extending along the lesser curvature nearly to the cardia. The pylorus would with difficulty admit a sound the size of a slate-pencil. The mass was exsected on the probability that it was cancerous, but the pathological report was that it consisted only of dense connective tissue without any trace whatever of malignancy.

Even cancer of the pylorus may run such an insidious course that it may be totally unsuspected until the sudden appearance of severe stenotic symptoms. In one of the writer's cases there were absolutely no symptoms whatever of impending disease until the sudden onset

of acute watery acid vomiting which continued in spite of abstinence from all food and liquids by mouth, the daily quantity vomited being about 5 pints during his starvation treatment. Operation on the third day showed a carcinoma the size of a lemon at the pylorus. The pylorus would not admit the passage of a lead-pencil.

Physical Signs.—Physical signs may be totally lacking in mild degrees of pyloric obstruction, even when the examination of the stomach shows an appreciable quantity of food residue in the fasting state. In more marked cases a variety of physical signs appear, the most important of which are due to hypertonus and increased peristalsis of the stomach wall.

1. Inspection may show the outline of the lower curvature of the stomach to be distinctly visible, provided the abdominal wall is not too thick. Inspection should be made by a strong and oblique light, and the line of shadow is best detected by standing behind the head of the patient as he lies on the table or couch for examination.

2. Waves of peristalsis may be observed from time to time passing in stately march over the region of the stomach from left to right and are most clearly in evidence several hours after the meal when the stomach is endeavoring to empty itself. The waves may be slight or they may be gross enough to be clearly visible at a distance of two or three yards, but feeble or vigorous they indicate a mechanical obstruction to the onward passage of food that is being compensated by more vigorous muscular contractions from behind. Absence of peristalsis does not exclude pyloric stenosis, as it may happen that the stomach is no longer able to rise to the emergency by contractions that are powerful enough to be visible. Such an enfeebled power may be the precursor of decompensation, for in these cases examination in the fasting state usually shows a rapid increase in the amount of residual food. The most vigorous peristalsis is seen with benign stenosis. In cancer the waves are usually feeble or may even be entirely absent, unless the cancer be engrafted on an ulcer that has already caused an appreciable degree of stenosis. The writer would regard the relative vigor of peristalsis as an important means of differentiating the benign and the malignant forms of the disease, although naturally too much reliance should never be placed on the presence of a single sign alone.

3. Peristalsis may be appreciated by palpation of the stomach area. At the time of vigorous contraction the organ may be distinctly felt as one might feel an inflated air cushion. This so-called "stomach-stiffening" may be more distinct at some parts of the stomach than at others, but usually it is recognized without difficulty, and like peristaltic waves is a sign of great diagnostic significance.

4. Succussion sounds and splashes are frequently heard during the palpation or percussion of the stomach, and are not of themselves significant of pyloric stenosis except when they are audible at the time in which the stomach should be empty. Succussion sounds readily elicited in the stomach before breakfast implies the presence of fluid in the fasting stomach, which ordinarily means pyloric stenosis.

5. Palpation of the pyloric region may or may not give evidence of disease. In making a systematic palpation of the organ it is generally best to map out the lower curvature and then to palpate along this line to the right and upward, so that the line of examination leads directly to the pylorus.

The presence of a tumor depends:

- (a) Upon the tonicity of the pyloric sphincter.
- (b) Upon the new tissue, cicatricial or malignant, that is deposited in or about the pylorus.
- (c) Upon the presence of extensive adhesions.

In stenosing ulcer that is not accompanied by the deposit of great amount of cicatricial tissue, there may be nothing that is palpable over the pylorus. Should spasm of the sphincter, however, be present it may be possible to feel the tonically contracted pylorus as a firm, short, cylindrical body which appears and disappears as the pylorus contracts and relaxes. At the time of relaxation a "pyloric squirt" may be audible through the stethoscope but is not of a diagnostic significance.

If the new tissue about the pylorus, whether cicatricial, tubercular, syphilitic, or carcinomatous, be sufficient to form a mass, a tumor becomes palpable, more or less movable according to whether or not it has become adherent to neighboring parts. Tumors of sufficient size to be distinctly palpable under ordinary conditions may be so drawn up under the liver by adhesions that it is not possible to detect them by palpation.

The characteristics of the tumor of cancer, ulcer, and of other forms of pathological infiltration are described in full under their respective headings.

6. To determine the size of the stomach and the degree to which it is dilated, a rough idea can be gained by locating the lowest point at which succussion sounds are heard: This is, however, a test which is inaccurate in many instances. The lower curvature may be mapped out by inspection during the period of gastric stiffening or by locating the lowest point at which peristaltic waves are visible. More accurate determinations are made by inflation, either by a bulb attached to a stomach tube or by effervescing powders.

By means of the x-rays the size and position of the stomach may be determined with great accuracy.

7. The mechanical weight of the stagnant contents tends to displace the stomach downward. If the pylorus be held up by adhesions the sagging is chiefly in the median zones and is of a sharply crescentic outline, but should the pylorus be freely movable the whole stomach not only sags downward but assumes more of an oblique position, so that the pylorus may be in the right iliac fossa and the greater curvature cross just above the pelvic brim.

It is interesting but not very important to know the exact measurements of the stomach. The important point to be decided is whether the stomach can do its work and empty itself as it should, and not how large the organ is or how distensible.

Diagnosis.—Gastric Analysis.—The diagnosis of pyloric obstruction may be made with absolute certainty by gastric analysis, while without this means of examination errors in diagnosis are apt to creep in. The most important examination by far is that of the fasting stomach. By the detection of appreciable amounts of fluid with or without food remains in the fasting state we have conclusive proof of a mechanical obstruction at the pylorus. To determine whether this obstruction be temporary, due to spasm, or permanent, due to structural change, repeated examinations may be necessary, and should by preference be made in all cases in which the disease is suspected. The amount of food-stasis and its resulting hypersecretion vary from time to time, so that the evidence afforded by the examination may be less conclusive at one time than at others. It not infrequently happens, especially in malignant cases, and even though the stenosis is quite marked, that the first examination may not reveal sufficient deviation from the normal to warrant any inferences, but that subsequent examinations may be such as to clearly demonstrate the presence of a mechanical obstruction of the outlet of the stomach. The importance, therefore, of repeated examinations cannot be too strongly emphasized. Functional disturbances of motility, such as atony, never lead to constant accumulation of liquid or food remains in the fasting state. *Food-stasis from atony does not occur.*

Gastric analysis may be identical in benign and malignant stenosis, while the examinations demonstrate the existence of a stenosis at the outlet of the stomach, they may not furnish any clue whatever to the exact nature of the obstruction. Unfortunately, this is often the case with cancer that is still in the operative stage. When gastric analysis points conclusively to a malignant origin for the disease, it is often too late to expect much from radical operations. Nevertheless, it is convenient from a clinical standpoint to contrast as best we may between the analyses in benign cases and those of malignant character.

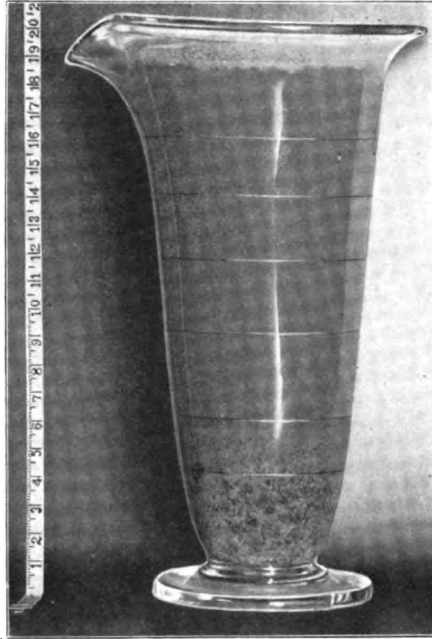
Method of Examination.—The patient should be directed to eat his dinner or his evening meal as usual the night before the examination. At 10 o'clock that night he should be told to eat a meat sandwich and to drink a glass of water, to which may be added a handful of raisins or a tablespoonful of dried currants. After this late evening meal he should take nothing by mouth, not even a sip of water, until he reports for examination the following morning. Should vomiting occur during the night the vomited matters should be brought for examination, and the time at which the vomiting occurred should be noted on the specimen. If several attacks of vomiting occur, the specimens should be separately collected and examined.

After such a test meal the stomach should normally be empty the following morning by 8 or 9 o'clock. Small quantities of acid fluid under 30 c.c. in volume may be disregarded. Quantities of acid fluid from 30 to 50 c.c. are on the borderline between pyloric spasm and pyloric stenosis of organic origin. Quantities exceeding 50 c.c. may occur in either conditions, but are rare with pyloric spasm. The nearer the quantity of fluid approaches 100 c.c. the more clear becomes the diagnosis of an organic obstruction.

Gastric Analysis in Benign Stenosis.—*Fasting Stomach.*—The quantity of supernatant liquids is usually more abundant than in the malignant form and shows a high acidity composed almost entirely of free and combined hydrochloric acid. The total acidity is almost always over 70, usually between 85 and 110, although it may be as high as 120. Reaction for free hydrochloric acid is sharp and decisive, and the total amount of the free acid may be represented by an acidity not more than 20 points less than the total acidity. It is, therefore, evident that the bulk of the fluid is composed of free hydrochloric acid, and that the combined acid is relatively less abundant. Slight reactions for lactic acid may occasionally be noted, depending upon the character of the food that has last been ingested, but positive reactions for lactic acid in appreciable amounts are not encountered in cases of benign stenosis.

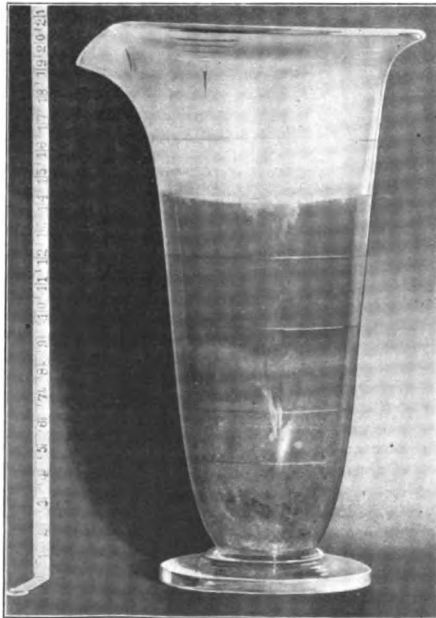
Sarcinæ are usually found and are characteristic of benign stenosis, as for their development not only does free hydrochloric acid seem necessary, but also a certain degree of stagnation of the gastric contents. When stasis exists and when the secretion of hydrochloric acid is maintained they are in their greatest development. When free hydrochloric acid fails, their growth may be maintained for a time at least by the loosely combined hydrochloric acid that may be present, but if after total disappearance of both free and combined hydrochloric acid, lactic acid formation gradually takes place, sarcinæ find the conditions hostile to their development and begin to disappear.

FIG. 68



Fasting contents in pyloric stenosis due to cicatrizing ulcer of the pylorus taken ten hours after the previous meal. Hypersecretion is here well demonstrated. Total acidity, 96; free hydrochloric acid, 74; no lactic acid; sarcinae present; no Oppler-Boas bacilli; blood positive.

FIG. 69



Fasting contents of benign pyloric obstruction ten hours after the previous meal. Total acidity, 80; free hydrochloric acid, 68; no lactic acid.

A few sarcinæ, according to Boas, may be found in the gastric contents of healthy persons, without being of diagnostic importance; it is only when numberless packets are seen in every field of vision that they are indicative of motor insufficiency of high degree. The writer has not as yet found sarcinæ in the contents of a normal stomach.

Sarcinæ are often present in the stools of those suffering from pyloric stenosis, especially in the evacuations of the gastrogenetic diarrhea that may complicate this disease. They are occasionally bile-tinged.

Yeast cells are often seen in abundance and show a proclivity toward the budding forms. Too much importance must not be placed on the finding of yeast, even of the budding forms, in the fasting stomach, as they may be found in apparently normal conditions.

Lactic acid and lactic acid bacilli are rarely found with the purely benign forms of pyloric stenosis and suggest malignancy.

Fermentation processes are less active in the benign forms than in the malignant, probably owing to the antifermentative power of the gastric juice, so that while organic acid may be present the quantity is rarely sufficient to give a characteristic odor to the contents. Carbohydrates if retained too long within the stomach may undergo decomposition, producing carbon dioxide, hydrogen, marsh gas, oil-forming gas, and other gases of fermentation, some of which are inflammable. The production of these gases is not inhibited by the presence even of large amounts of hydrochloric acid. The mechanical obstruction to the digestion is the chief cause for the fermentation; the presence or absence of hydrochloric acid is of inferior importance.

Decomposition of the albumin may later take place in the ectatic stomach, even in the presence of free hydrochloric acid, and one of its products, sulphuretted hydrogen, can be readily detected.

Three grades of severity of the disease may be recognized, according to the gross appearance of the fasting contents.

In the mild form we find a fluid of clear or slightly opalescent appearance, which on standing shows a slight sediment, the constituents of which cannot be determined by the naked eye, but which usually under the microscope prove to be finely digested food remains, usually of the carbohydrate group of foods. The quantity varies from 30 to 40 c.c. to 100 or even 120 c.c. This mild form is usually described as chronic hypersecretion or Reichmann's disease.

In the severer cases we have a difference in degree only. The quantity of fluid is somewhat more abundant, the sedimentary layer more clearly defined, and it becomes evident to the naked eye that the sediment is composed of food remains distinctly to be recognized as such. In these milder cases fermentative changes are rarely present.

In the most severe cases the quantity taken from the fasting stomach

is strikingly copious and may even amount to one or more liters. In one of the writer's cases 11 pints of fluid and of food remains were withdrawn on one examination.

On standing, three layers are more or less clearly defined. The lowest consists of food remains of recent or of ancient date, more or less perfectly digested, and often fermenting. The depth of this sedimentary layer represents the actual amount of permanent food-stasis.

The middle layer consists of slightly turbid or opalescent fluid, varying in amount according to the degree of the hypersecretion. The fluid layer may be brownish from altered blood, or in exceptional instances may be bile-tinged. Bile may thus be present even with demonstrable stenosis of the pylorus in cases of infiltration of the pyloric wall which lead to a "rigid patency" of the outlet. When such a condition occurs, the pyloric sphincter may be unable to relax sufficiently for the normal passage of chyme through it, and at the same time be quite unable to contract and prevent the regurgitation of bile, especially if straining efforts be made by the patient during the passage of the tube.

The upper layer is of comparatively shallow depth and consists of mucus, swallowed saliva, and pharyngeal secretions intermixed with gross particles of food that are light enough to float, often frothy from gases of fermentation.

In the milder cases, when the bulk of the contents consists chiefly of surplus gastric juice, no abnormal odor may be detected. When food-stasis is more pronounced the odor may be sour, yeasty or rancid according to the amount of organic acids generated by fermentation.

Sulphuretted hydrogen may be detected in both benign and malignant stenosis, but is more common with the latter group. The presence of the gas may be easily demonstrated by any of the chemical tests. A simple test is to place the contents in a tightly corked bottle, in which pieces of filter paper moistened by a solution of lead acetate and caustic potash are suspended from the cork. Odors that are foul or putrid suggest ulcerating neplasms.

Test Breakfast in Benign Stenosis.—The examination by the test breakfast should follow immediately after the fasting stomach has been emptied by aspiration. No attempt should be made to wash the stomach nor to introduce any water through the tube. After the fasting stomach contents have been aspirated, a roll and a glass of water should be given and the products of digestion removed at the expiration of an hour.

The test breakfast may or may not be indicative of pyloric stenosis, and compared with the examination of the fasting stomach is of inferior importance. It may happen that aspiration in the fasting state with-

draws a quantity of acid fluid with a sediment of food remains, scanty, but quite sufficient to be diagnostic, while the test breakfast of such a case examined after the fasting test may show only a moderate degree of alimentary hypersecretion; the total quantity aspirated may exceed the normal, and the supernatant liquid on standing may be far greater in depth than that of the sedimentary layer, but the few vestiges of older food remains that would establish the diagnosis are lost amid the greater quantity of breadstuffs of which the test breakfast is composed. Ancient food remains are easily overlooked in test breakfasts unless present in fair amounts and distinguishable from the digested breadstuffs by color, form, or outward appearance. Numberless errors of diagnosis are made because the test breakfast is alone examined and no investigation whatever is made of the contents in the fasting state. Unless the fasting examination had been made the condition of food-stasis might have been completely overlooked.

Benign stenosis is regularly accompanied by an alimentary hypersecretion. The test breakfast is abundant in quantity and more fluid in consistency than normal, ranging from 200 c.c. to 300 c.c., but occasionally being even more copious. Quantities of test breakfast exceeding one-half liters are not uncommon.

In mild forms two layers form on standing, the layer of supernatant fluid being more than equal in depth than that of the lower sedimentary deposit of well-digested breadstuffs with a probable addition of older food remains. In the majority of cases the proportion between the two layers is 2 to 1 or 3 to 1, but exceptional proportions of even 10 to 1 are encountered. In severe types three layers are defined as in the fasting contents, a lower sediment of new and old food residue, a middle or liquid zone, and a floating layer of food, saliva, mucus, occasionally frothy.

In certain cases of benign stenosis total acidity may not be abnormally high, attaining only the normal, or in exceptional instances being far under the normal. In other instances the total acidity may be normal or over the normal, while the acidity that is due to free hydrochloric acid may be decidedly subnormal. It may be demonstrated that in these cases the greater part of the total acidity is composed of loosely combined hydrochloric acid.

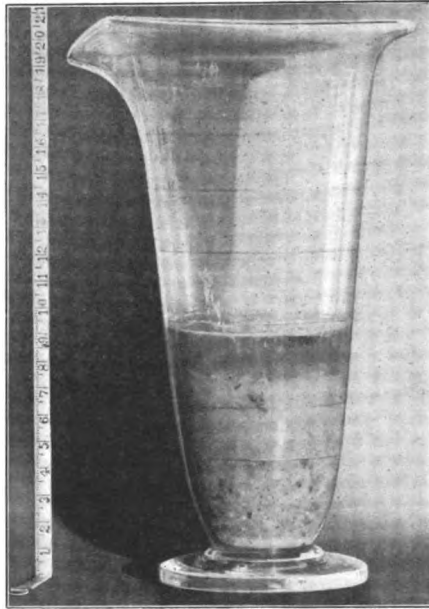
Hyperacidity is not a necessary accompaniment of benign stenosis. Hypersecretion is, on the other hand, of great diagnostic importance.

Gastric Analysis in Malignant Stenosis.—This is that observed with cancer of the stomach associated with food-stasis.

Fasting Stomach in Malignant Stenosis.—The fasting stomach regularly reveals evidences of food-stasis with more or less hypersecretion of a fluid that may consist of hydrochloric acid alone, of hydrochloric acid

with lactic acid or of lactic acid alone. The contents are often exceedingly offensive, foul, or even fetid or putrid. The ordinary tests for occult blood are usually quite well-marked. Sarcinæ are rarely present, except in the transition form of cancer engrafted upon ulcer, but Oppler-Boas bacilli are almost regularly present in stagnant achlorhydric gastric contents containing lactic acid. The diagnostic importance of the Oppler-Boas bacilli coincides essentially with the presence of lactic acid itself, and their detection is only of real value if they are found in a specimen which gives a doubtful lactic acid reaction.

FIG. 70



Fasting contents of pyloric stenosis due to cancer. Total acidity, 24; free hydrochloric acid, 0; lactic acid marked; Oppler-Boas bacilli present; blood positive.

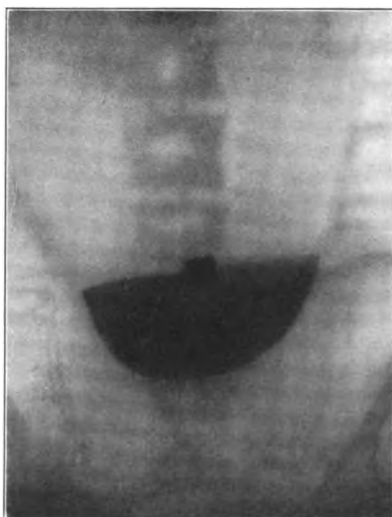
Test Breakfast in Malignant Stenosis.—The test breakfast shows the characteristic chemical and microscopical examination of cancer of the stomach generally, *plus* food-stasis and a tendency toward hypersecretion. The filtered fluid may contain free hydrochloric acid alone or lactic acid alone, or neither form of acid may be present. Positive reactions for both hydrochloric and lactic acids are more constantly present than in the benign forms, and the odor is more offensive. Blood is almost invariably present in either occult or visible form, though delicate tests may be required before positive reactions are obtained.

There is hardly any doubt possible as to the nature of the stenosis

when lactic acid is present in stagnant gastric contents, but there are a large number of cases in which lactic acid and other suggestive signs of cancer are absent, in which the *analysis is identical with that of ulcer, so that a differential diagnosis by gastric analysis is totally impossible.*

Gastric Analysis of Cases of Transition of Ulcer of the Pylorus into Cancer.—Beginning in the early stages with the gastric analysis indicative of ulcer—food-stasis, hypersecretion, and hyperacidity—there is a gradual tendency toward a reduction in hydrochloric acid, and a formation of lactic acid. Sarcinæ hitherto present disappear and are replaced by the lactic acid bacilli. The analyses now become characteristic of the malignant form. The early cases cannot be told by gastric analysis alone from ulcer—the ultimate analyses merge into those of cancer. It is only the intermediate cases, in which both hydrochloric acid and lactic acid are present, that are clearly indicative of malignant degeneration of chronic gastric ulcer.

FIG. 71



Pyloric stenosis: benign bowl-shaped residue after six hours. (Radiologist, Dr. Leaming.)

Radiographic Diagnosis.—The Röntgen picture of pyloric stenosis is quite characteristic. On the plate taken six hours after the first bismuth meal there is a large bowl-shaped residue of bismuth, which extends far to the right of the navel. The amount of bismuth residue is far greater than in simple atony. Radiographs of the stomach taken immediately after the second bismuth suspension meal usually show a large well-filled stomach, the greater curvature of which sags downward and to the right, passing thence upward and to the left to the

pylorus, giving to this portion of the stomach the "undershot" appearance of a bull-dog's jaw. Peristalsis may be more than ordinarily active.

For the differences in the radiographic findings of ulcer with or without adhesions, and of cancer, the reader is referred to these respective headings. The radiographic diagnosis of stenosis is much inferior to the simpler and more positive evidence of finding food remains in the fasting stomach with the tube.

Diagnosis in General.—The diagnosis of pyloric stenosis can easily be made by repeated examinations of the fasting stomach, but the diagnosis should not rest with that of the pyloric stenosis alone, without an attempt to ascertain whether the obstruction be organic or partially or wholly due to spasm or tumefaction, and furthermore, to decide, if possible, whether we are dealing with the benign or with the malignant form. Gastric analyses should be made at intervals throughout the entire course of benign stenosis, so as to detect as early as possible any deviations that might indicate beginning malignancy.

1. The extent of the constriction that is caused by spasm or congestive swelling is to be determined by the intermitting or remitting severity of the symptoms, by repeated examinations of the fasting stomach and to a limited extent by the results of treatment. The diet and mode of life remaining the same and the conditions of the test being uniform, variations in the amount of the residual contents indicate that one or both of these remediable factors are at work, although the conclusions may not be accurate in all cases. It is conceivable that with a fixed degree of obstruction in front, varying degrees of muscular efficiency behind will allow of great variations in the amount of propulsive work actually performed, owing perhaps to daily variations in the nervous strength of the motor impulses conveyed to the stomach walls. It is always advisable unless the condition call for immediate surgical relief to estimate the variations in the amount of the residual contents by repeated tests and to attribute the minimum quantity of residue to organic contracture, and the difference between the minimum and the maximum quantity of retained contents to the effect of temporary factors. To remove these latter causes for an increase in the mechanical obstruction, is the one object of our medical treatment. Time should not, however, be spent in pottering with tests when malignancy is suspected.

2. To distinguish clinically between benign stenosis of the pylorus and that produced by malignant neoplasm is not as simple a matter as it may appear. Classical examples are encountered in which the differential diagnosis is obvious and could be made by those even of limited experience and powers of observation, while, on the other hand, the symptoms, physical signs, and gastric analyses in both forms may be

identical. Moreover, the symptoms of stenosis of undoubted benign origin may gradually and almost imperceptibly merge into those of malignancy from the slow development of cancer on this previous ulcer base.

It is impossible therefore to contrast the points of differential diagnosis in any table of parallel columns, for a table so constructed cannot endure the test of experience.

It is said that a past history of ulcer is indicative of benign stenosis; but malignant changes may develop on the ulcer base, and a case manifestly malignant may give a long antecedent history of chronic ulcer, so that the evidence afforded by the history is faulty and misleading. A sudden onset and a progressive course may be attributed to cancer, and yet chronic obstruction by cicatricial contraction may remain latent until the sudden appearance of stenotic symptoms which run a progressive course, while on the other hand early carcinoma, especially if engrafted on an ulcer base, may give a slow insidious onset and the symptoms may remain practically stationary for months.

Gastric analysis often proves misleading, except that lactic acid, Oppler-Boas bacilli, and evidences of ulceration in the gastric contents point to malignancy. The combination of hydrochloric and lactic acids with either sarcinæ or lactic acid bacilli in a patient with an ulcer history indicates carcinomatous degeneration of the ulcer with sufficient distinctness to warrant an exploration.

Each case has therefore to be decided on its own merits, and every point in relation to the case carefully considered. The age of the patient, his previous history, the subjective symptoms and physical signs, the presence or absence of metastases, the results of repeated gastric analysis, the question of an advancing chloranemia or cachexia should all be weighed in the balance and a verdict given only when all evidence is carefully and judicially sifted.

From a practical standpoint the advice and moral support of a competent conservative surgeon should be requested, and in cases of doubt an exploration is generally advisable.

Prognosis.—The prognosis is that of the disease of which pyloric stenosis is a resulting complication. In benign stenosis the prognosis should be that of the operation done for its relief, and should not exceed a mortality of over 2 or 3 per cent. for simple gastrojejunostomy provided that the operation is performed by a competent surgeon with some experience in abdominal surgery. A higher risk attends the more radical operation of exsection. Naturally the operative risk is less when the operation is performed at a time when the patient is in comparatively good health than when operation is resorted to as affording the only chance for life at a time when the patient is practically dying of starvation.

Many untreated cases may give a history extending over years, either steady or remittent. If the lesion be extreme the course is steadily downward and is attended by progressive weakness, emaciation, and slow starvation. In these advanced cases vomiting may become quite infrequent, and may occur only after intervals of several days or even a week; but when it does there are vomited such large quantities of stagnating fermenting material, "pailsful," as the patient may express it, that it is quite evident that there is represented the accumulation of days.

If during the course of chronic pyloric stenosis acute exacerbations occur with copious watery vomiting, even in spite of total abstinence from all food and drink, the symptoms may become urgent in the extreme, and if unrelieved by treatment or by timely surgical intervention may result fatally within a few days.

Tetany was formerly regarded as a complication practically hopeless, the mortality of the earlier cases amounting to 90 per cent. Improvement in medical treatment reduced this frightful fatality to 70 per cent. Recently, however, the reports of cases treated surgically have been coming in, and show conclusively that the hope of recovery lies in the surgical treatment of the disease.

The latest figures available are those of McKendrik,¹ who has collected 24 operative cases of gastric tetany with but 3 deaths, a mortality of 12.5 per cent. Of the 3 fatal cases death was due to visceral disease, to pneumonia, and to peritonitis.

In cancer the prognosis depends upon the possibility of radical removal of the neoplasm. The operative risk of all operations for cancer of the stomach, whether radical or palliative, is considerably higher than in similar operations done for the relief of benign obstruction.

Treatment.—Although theoretically the treatment of pyloric stenosis should be surgical, nevertheless there are mild cases which get along very well by medical means alone, and which improve to such an extent that operative interference is quite unnecessary.

For those who are about to undergo an operation for relief, a medical course of treatment is almost invariably indicated, by which the stomach may be emptied of its stagnating contents and cleansed, and the general strength and nutrition so improved that the patient may be better able to withstand the surgical ordeal that confronts him.

The object of the medical treatment is to reduce if possible the degree of the stenosis by relieving concomitant pyloric spasm and tumefaction, as well as to feed the patient and improve his nutrition. Means of medical relief are afforded by diet, by lavage, and by drugs.

¹ Scottish Medical and Surgical Journal, 1907, xxi, 253.

The results of medical care are better shown by a diminishing amount of residual food remains, and by a gain in weight, than by any apparent improvement in the patient's comfort or sense of well-being. Beneficial effects of treatment are demonstrated by the clear fluid of hypersecretion being found in the fasting stomach instead of macroscopical food remains.

Dietetic Treatment.—The most important consideration is the avoidance of any food that may resist digestion and persist as gross food particles too large to readily pass the constricted pylorus. The food should be finely comminuted, thoroughly masticated, and should not contain large indigestible particles, such as grape-skins, prunes, or tough and gristly portions of meat. Food that is mechanically irritating, such as coarse vegetables, may increase the liability to pyloric spasm.

The quality of the food depends largely on the digestive power of the stomach. In benign stenosis, when the secretions are rich in hydrochloric acid and of good peptic power, the quality of the food is quite unimportant. Should, however, hydrochloric acid be absent, as frequently occurs, with malignant stenosis, the proportion of nitrogenous food should be reduced and food rich in carbohydrates should be correspondingly increased. Although we may attempt theoretically to regulate the diet according to the secretion of the stomach, practically we are often forced to disregard these theoretical considerations and to arrange the diet the best we can according to the desires of the patient and his ability to carry out the dietetic treatment proposed.

More important than the quality of the food is the quantity which may be taken at any one meal. Pyloric stenosis is an indigestion of quantities, and the invariable rule should be to insist on frequent small meals, so that the burden of the food is divided throughout the day. At least five or six meals should be taken daily. A great deal of nourishment can be given in the twenty-four hours without overloading the stomach at any one time and without causing distress to the patient, whereas a single large meal will be followed by pain and distress, often so severe that the patient bitterly regrets his indiscretion.

Liquids should be restricted at the time of the meals, but may be given between meals, although in small quantities at a time. Unless it is evident that the pylorus is impervious to fluids and the stomach is distended by liquid contents, fluids may be sipped throughout the day so that a total sufficient quantity is absorbed.

It has been recommended that the diet should be concentrated so that mechanical distention of the stomach does not result from mere bulk. Scraped meats, eggs, concentrated solutions of peptone, and the like are examples of the food advocated according to this

principle. An opposed view is that the diet should be entirely liquid so that the nourishment may more readily pass through the narrow outlet. The basis of such a treatment is milk. The writer has found it impractical to adhere to either of these extreme forms of diet; his preference is decidedly for solid or semisolid foods, finely comminuted or in purée form, so that there are no gross food particles sufficient in size to occlude the pylorus. Milk should never be used unless thoroughly peptonized, as coagula are difficult of digestion and often too large to escape through the pylorus. Milk as ordinarily given is one of the worst forms of food that can possibly be devised for these patients. If milk be peptonized the warm process is better than the cool. To peptonize the milk completely, the author's method is as follows:

To a pint of milk add $\frac{1}{4}$ pint of water. Divide the milk into 2 equal parts, boil one and to it add the other. The contents of one Fairchild's peptonizing tube is then to be added and the whole of amount of milk is to be placed in stoppered bottles well immersed in water that is distinctly warm to the hand for one and one-fourth hours. The milk is then rapidly boiled and placed on ice.

A simple diet for advanced pyloric stenosis may be arranged as follows:

- 8 A.M. One-half cup of cocoa, soft-boiled egg with fine cracker crumbs.
- 10.30 A.M. Scraped beef sandwich with bouillon.
- 1 P.M. Rather thick purée of vegetable, such as pea or bean soup, a fine cereal, as hominy or cream of wheat, with cream and sugar.
- 3.30 P.M. Malted milk.
- 7 P.M. Creamed toast, baked custard.

In less severe cases the diet may be somewhat more varied, although it should follow the general principles previously indicated. The heaviest meal should preferably be at mid-day, and the evening supper should be light so as to avoid nocturnal distress. Such a diet may be outlined as follows:

- 8 A.M. Cup of cocoa, two soft-boiled eggs, fine cereal.
- 11 A.M. Malted milk.
- 1 P.M. Purée of vegetable soup, creamed fresh fish, or finely chopped creamed chicken, mashed potato, or any vegetable that has been passed through a purée sieve, such as turnips, carrots, etc. Corn-starch pudding.
- 4 P.M. Cup custard.
- 7 P.M. Fine cereal, scraped beef sandwich or poached eggs on soft toast, junket, tapioca or rice pudding.

If it can be demonstrated that not enough fluid is being absorbed by the system, that the urine and blood are concentrated, and the tissues are in an abnormally desiccated condition, fluids should be

introduced into the system in every possible way. In these cases there is very little use in forcing fluids by mouth. The Murphy drip should be used and as much water as possible induced by the rectum. The writer prefers normal solutions of sodium citrate or of sodium bicarbonate to the salt solution ordinarily employed. Glucose up to 3 per cent. solution may be used in combination with the citrate, and has the advantage of adding nourishment of high caloric value to the fluid. In desiccated subjects on whom an operation is advisable, fluids should be introduced into the system in large amounts prior to the operation. If haste is indicated, hypodermoclysis may be employed.

Treatment by Lavage.—The stomach should be usually washed every day before breakfast, so that the patient starts the day with a clean stomach. The morning lavage has this advantage that we are not so likely to be washing out from the stomach nourishment that might pass the pylorus and become absorbed, and moreover, the stagnant contents found in the stomach in the morning are really quite unfit for propulsion into the intestine for absorption. For those whose stomachs fill up during the day and cause nocturnal distress and vomiting, lavage may be recommended either before dinner or at bedtime. Washing the stomach before the evening meal empties it of its previous accumulation so that the last meal of the day finds conditions most favorable for its easy passage and absorption. Lavage the last thing at night will naturally benefit distress and vomiting, but has the disadvantage of washing out nourishment that might be later utilized. It is, therefore, not recommended as a routine procedure to be used over any considerable length of time, but may be resumed from time to time, especially during acute exacerbations of the ailment, to insure for the patient a good night's rest.

In very advanced cases in which the stagnant stomach contents are abundant and fermenting, it is better to empty the stomach every morning, and if necessary before the evening meal as well, by aspiration before introducing any liquid into the stomach through the tube, as it may happen that the sudden washing of the stomach may be followed by gastric tetany, the reason for the occurrence being that the toxins are dissolved in the lavage water so quickly that they pass more readily through the pylorus and are quickly absorbed.

Lavage in stenosis is usually simple. The inflow of water is slow, but the return is usually rapid and forcible, so that the residual fluid after the lavage is generally small in amount compared with normal cases. The simple tube and funnel are therefore usually sufficient provided that the tube be of sufficient caliber and the eyes are large enough to readily admit large food products. The ordinary

small caliber of tubes in the market is totally inadequate for the purpose, as it becomes so easily blocked. One might as well wash the stomach through a catheter as through many of the tubes commonly employed.

For office lavage the writer's apparatus, described on p. 68, is to be recommended. Lavage should be continued at each sitting until the stomach washes clean, even though this may take considerable time at first. After the stomach has once been thoroughly cleaned and kept clean for a time lavage becomes easier and easier. At first the washing of the stomach should be under the personal supervision of the physician; it is only when the patient becomes an adept that he may be intrusted to carry out his own treatment.

The writer's preference is to wash with plain water. The object of the lavage is to empty the stomach of its contents and this can be done with plain water as well as medicated solutions, with the great advantage that we obviate the danger of poisoning should large quantities of residual water be retained. If the contents of the stomach are foul or fermenting, the writer recommends adding sufficient essence of peppermint to the lavage water to give it an agreeable odor and taste. After such lavage patients feel cleaner and more comfortable than they do after plain water has been used. Bicarbonate of soda may be added, a dram to a pint, in cases with high hydrochloric acidity. Among the various forms of medication that have been recommended are resorcin in 1 per cent. solution; salicylic acid in $\frac{1}{2}$ per cent. solution; thymol in $\frac{1}{2}$ per cent. solution; ichthyol, 20 to 30 drops to a quart; creolin or lysol, 10 drops to a quart, or sodium benzoate in a 1 to 2 per cent. solution.

If any of these forms of medication are used the return flow from the stomach must be measured and compared with the amount of medicated water introduced, so as to obviate the possibility of retention and absorption of these drugs.

Drug Treatment.—Oil before meals is often of service in reducing pyloric spasm. Plain olive oil may be given in $\frac{1}{2}$ to 2-ounce doses before meals or a single large dose of 2 or 3 ounces may be taken on retiring. A 3 per cent. solution of anesthesin in oil may be given in $\frac{1}{2}$ to 1-ounce doses before meals, and often proves highly beneficial in reducing a possible spasmodic element of the stenosis. Oil may also be given through the tube at the completion of lavage, 2 to 4 ounces of the warmed oil being introduced in this manner.

Cohnheim is an enthusiastic advocate of the oil treatment, and states that in spastic stenosis that an absolute cure will follow, while in cicatricial stenosis more relief is obtained than after any other method of treatment. He recommends the washing of the stomach

in the morning and the induction through the tube of 100 to 150 c.c. of warmed oil. After this the patient lies on the right side and fasts for one hour. If the pains do not disappear during the day 50 c.c. are taken before retiring. Later in the treatment a wineglassful an hour before breakfast and 1 or 2 tablespoonfuls one or two hours before dinner and supper will suffice in mild cases. Emulsion of almonds may be substituted for the last two doses.

Belladonna or atropine are often of service in reducing pyloric spasm and may be given a trial. The writer's results, however, have not been satisfactory with this drug, except in a few cases of sudden complicating pylorospasm, with the constant vomiting of acid fluid. In the slow, ordinary cases of pyloric stenosis no good has apparently followed this medication, while, on the other hand, the thirst and dryness of the mouth have been intensified. The following is the form in which the drug has usually been given and may be recommended for trial:

R—Tinct. belladonna	3ij
Chloral hydrate	3j
Resorein resubl. (Merck)	3j
Strontii bromid.	3iiss
Aq. chloroform.	3iv
Spirits anisi	gtt. viij

M. Sig.—Teaspoonful in a wineglass of water every three hours.

When hypersecretion exists with a high degree of acidity, alkalies are of use in reducing the acidity and making the patient much more comfortable. According to the observations of Hertz and others the higher the acidity the greater are the peristaltic contractions of the stomach wall. A reduction of the acidity will therefore reduce the pain and discomfort and may minimize pylorospasm. When alkalies are given they should be given in sufficient quantities to do the work. One or two teaspoonfuls of bicarbonate of soda may be required at a single dose, if the gastric contents be copious and very highly acid.

In pyloric stenosis treatment by mineral water is distinctly contra-indicated. The stomach has enough to do without adding to its burden large amounts of water that are recommended at the medicinal springs. Thiosinamine has been superseded by an analogous preparation, fibrolysin, as a possible softener for cicatricial tissue. Fibrolysin may be obtained in sterile form ready for use in glass bulbs, each bulb containing 2.3 c.c. of a solution of $1\frac{1}{2}$ parts fibrolysin to $8\frac{1}{2}$ parts distilled water (Merck). Each bulb contains a single dose for hypodermic injection, and should be given into the gluteal muscle as injections into the skin may be followed by necrosis. Any untoward effect of fibrolysin may be obviated by stopping the treatment as soon

as the odor of onions appears upon the breath. The author has had no experience with the preparation, as the reports of cases in which it has been used have been exceedingly disappointing.

Strychnine has been recommended to increase the peristaltic power of the stomach so as to force its contents through the constructed outlet. Heavy massage of the stomach from left to right has been recommended for the same purpose. For the stimulation of peristalsis, hydrotherapy has also been recommended in the form of cold needle douches, cold showers, and alternating hot and cold needle sprays. In cases of pyloric obstruction in which peristalsis is visible and evident, there seems to be but little use for measures whose object it is further to increase peristalsis. The muscular power of the stomach is good enough; the trouble lies in the resistance in front, and the writer does not believe in increasing peristalsis artificially, as it is unwise to overwork the muscular power of an organ that is doing its best. For this reason the writer believes that strychnine, hydrotherapy, and forcible massage are positively contraindicated in this disease.

Hot compresses are, however, serviceable as a means of reducing pyloric spasm. The electric pad may be applied, or the warm, moist compresses commonly in use. These have been described under the treatment of ulcer, p. 173. Relief from symptoms often follow recumbency, and the patients learn for themselves that they feel better when they lie down after meals. Complete rest in bed for several days or a week at a time is often of service, and should be recommended in severe cases. In many instances rest in bed is essential to improvement.

Operative Treatment.—In benign cases of stenosis that are slight and stationary, the patient may go on for years quite comfortable and content, provided he follow out the proper diet and wash his stomach from time to time. Although the curative treatment of these patients is surgical, nevertheless as long as they do as well as they seem to be doing it is not common-sense to operate on them. Operation is indicated by any of the following conditions:

1. Operation is indicated if macroscopical food remains be constantly present in the fasting stomach after medical treatment has been carried out faithfully for a reasonable period, especially if the quantity of the fasting stomach contents show a tendency to increase, indicating that the lesion is a progressive one. Operation is naturally more frequently advised in young individuals who otherwise would have to spend their life under medical treatment, than in those of advanced years who are content to get along as they are, under medical care. The operative risk is naturally much greater in aged patients.

2. Operation is indicated if the lesion be so extreme that not enough food can pass into the bowel to keep the patient well nourished.

Surgery is indicated whenever there is a progressive loss of weight under medical treatment.

- 3. Operation is indicated if the symptoms of pain and vomiting continue in spite of treatment.

4. Operation is indicated should acute exacerbations of the ailment occur, characterized by profuse vomiting of acid fluid. In these cases it is a matter of nice judgment when to operate. If we wait too long the patients are exhausted from vomiting and by their loss of fluid. It is better to operate too early than too late upon these cases. If, after interdiction of all food and fluids by mouth the vomiting continue more than forty-eight hours, the patient should receive fluid by skin and rectum as quickly as possible, and an operation done at the first advisable moment.

5. Gastric tetany is a cause for immediate operation, and the operation should invariably be preceded by or accompanied with hypodermoclysis and the induction of water into the rectum by the Murphy drip. Calcium lactate should be given in gr. xv dose every three hours by mouth, or gr. xx every three hours by rectum.

Surgery is indicated whenever there is the least suspicion of malignancy, and exploration should be performed without loss of time so that a radical operation may, if possible, be done.

Painstaking examination should be made in these cases for any indications of inoperability, such as enlarged cervical glands or implantation in the vesicorectal pouch. If a radical operation be deemed impossible, the palliative operation of gastrojejunostomy may be postponed until the pyloric stenosis reaches a point when the patient's nutrition begins to fail, or when the symptoms become distressing. In these cases it is not wise to wait too long, for then we submit a patient who is debilitated by disease to a severe operation. Gastrojejunostomy in malignant stenosis may be followed by most brilliant results, a cessation of pain and vomiting, and by a gain in nutrition that is often most striking.

The patient may feel comparatively well, and eat almost anything with impunity, and such a period of improvement may continue for some months after the operation before the symptoms reappear. The downward course of the disease is then usually rapid.

CHAPTER XIII

CONGENITAL PYLORIC STENOSIS

INFANTILE PYLORIC STENOSIS

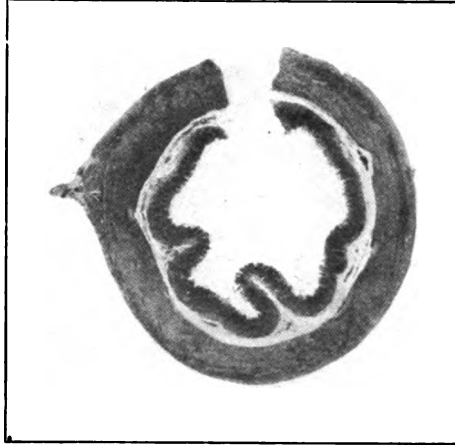
• PYLORIC stenosis occurring during infancy has been recognized as a clinical entity for many years, but it is only since the attention of the profession was called to this ailment by Hirschsprung of Copenhagen, in 1888, that the disease has been universally recognized. The number of recorded cases after Hirschsprung's paper was at first not at all numerous, but guided by the accurate description of the symptoms of the condition by later authorities, more and more cases have been correctly diagnosticated, and the additions to the literature of the subject have become more numerous.

Pathology.—The essential lesion is the thickening of the pyloric ring from hyperplasia of the circular muscular fibers. The pyloric canal assumes a funnel-shaped or a cylindrical form, hard and incompressible. On section its walls are thick and dense. The pyloric orifice may be so contracted that only the passage of a fine probe is possible. The duodenal end of the pylorus projects into the duodenum, and suggests the appearance of the cervix uteri projecting into the vagina. The mucous membrane of the pyloric canal shows usually one large, single, longitudinal reduplication with many smaller parallel folds. This longitudinal reduplication or projection of the mucous membrane still further increases the stenosis. The muscular hyperplasia fades gradually away on the stomach side of the pylorus, but terminates rather sharply at the pyloric ring, although it is possible for muscular hypertrophy to extend outward a short distance into the duodenum. The longitudinal fibers are but little affected, although in a few instances this muscular layer shares to a small degree in the hyperplastic process. The stomach itself is usually dilated. Its wall in the pyloric half shows evident muscular hypertrophy, which fades away toward the fundus so that the wall of this latter portion of the stomach is of normal thickness, or may even be thin.

Etiology.—There are two distinct theories, each supported by competent and experienced clinicians and pathologists to account for this interesting condition. Each theory is supported by facts which seem reasonable and plausible, but any argument for one or the other theory

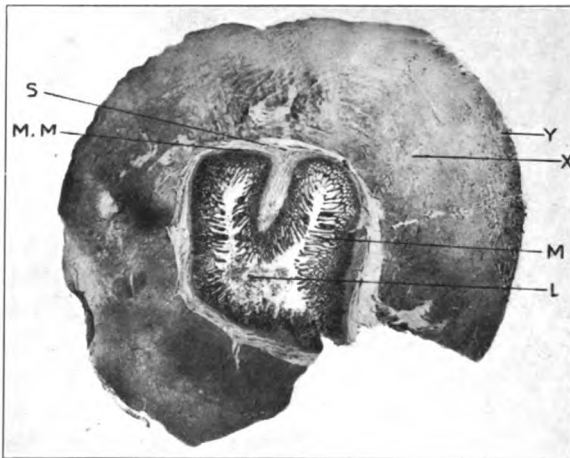
may be opposed by an equally good argument against it, so that at the present time judgment as to the true nature of infantile stenosis must

FIG. 72



Transverse section of normal pylorus of an infant. Compare with photomicrograph of congenital stenosis. (Shaw and Ordway, *American Journal of Diseases of Children*, September, 1911 vol. ii)

FIG. 73



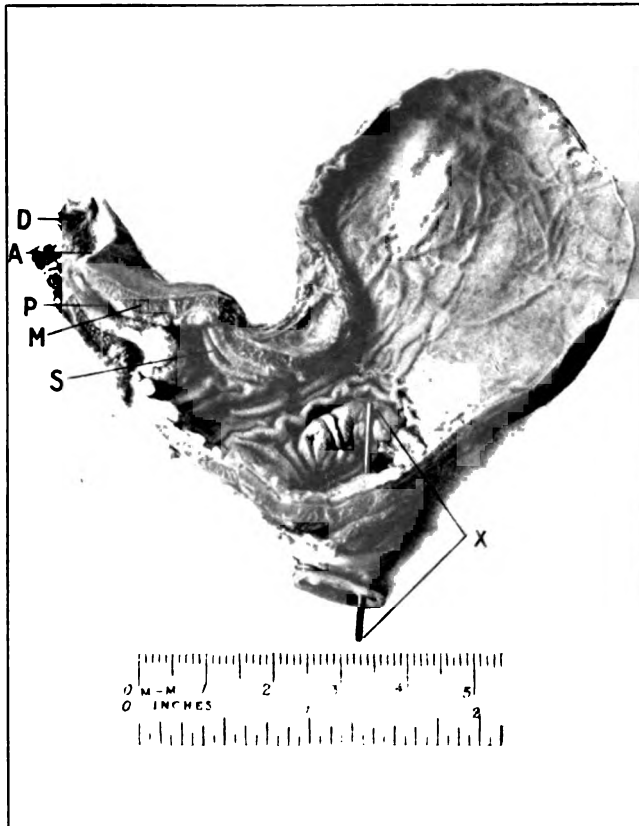
Congenital hypertrophic stenosis of pylorus. *L*, lumen of pylorus much contracted; *M*, mucosa; *M, M*, muscularis mucosæ; *S*, submucosa; *X*, very much hypertrophied circular muscular coat. At the outer edge of this coat one can see, as at *Y*, narrow strips of the longitudinal coat. (From the Pathological Laboratory of the Babies Hospital.)

be withheld. A study of the cases in literature, however, lead us to infer that there are two distinct pathological conditions which give identical physical signs and symptoms, one of which is amenable to

medical treatment and the other progresses toward a fatal termination unless relieved by surgical operation.

The first theory is that the muscular hyperplasia of the circular fibers surrounding the pyloric canal is an error in development. The pyloric sphincter is essential for the proper mechanism of digestion, and

FIG. 74



Congenital hypertrophic stenosis of the pylorus on which gastro-enterostomy was performed. Gastro-enterostomy opening through which a glass rod is inserted is seen at X. The pyloric canal (P) is very markedly narrowed, the walls being in close apposition. The very marked hypertrophy of the circular coat of the muscularis with its fibrous tissue septa is seen at M. Just outside of this the longitudinal muscular coat may be seen. The submucosa (S) also shows hypertrophy. A, pyloric valve; D, duodenum. (From the Laboratory of the Babies Hospital.)

nature in these cases has simply overdone the matter and has supplied muscular tissue far in excess of the quantity required.

Those who believe in this theory find an apparent corroboration in the fact that infants dying early in the course of the disease show too much muscular tissue for it to have been formed after birth by a process

of compensatory hypertrophy. Those who oppose this theory do so on the ground that were the process truly congenital evidence of this error in development would occasionally be found in the fetus, whereas the fetal hyperplasia of the pyloric musculature is almost unknown, only one case having been recorded.

The second theory is that there occurs a primary spasm at the pylorus to overcome the resistance of which a muscular hypertrophy takes place. This spasm may be of intra-uterine origin and probably results from disturbances of coördination in the motility of the fetal stomach, or the spasm may occur after birth from fissure or erosion of the pylorus or duodenum. Still has advanced an ingenious theory that the spasm is the result of "stomach stuttering"—a disturbance of muscular coördination akin to the stuttering so common in children who are learning to talk.

Küttner¹ reports 2 cases of duodenal ulcer in infants, one babe being but four days old, probably embolic in origin from thrombosis of the umbilical vein, and speaks of the possibility of many cases of infantile pyloric stenosis being due to the spasm thus induced. He quotes a case of von Torda's, in which an infant, aged eight months, died with a clinical picture of congenital pyloric stenosis, and in which the cause for the condition was found to be an ulcer of the duodenum 5 mm. below the pyloric ring.

Those who believe that the lesion is a muscular hypertrophy resulting from spasm meet the argument that such hypertrophy requires more time for its development than seems possible in the cases that die early in the disease, by saying that one cannot reason from the development of the adult to that of the infant and about the rate of development of hypertrophy in infants we know practically nothing.

Cautley² regards the evidence that hypertrophy results from prolonged muscular spasm as quite unconvincing. There is no proof of hypertrophy following spasm in other ages, and there is no reason why it should follow in infancy and not at other times in life. Prolonged anal spasm does not cause hypertrophy of the rectal sphincter. Hypertrophy, moreover, does not disappear after gastro-jejunosotomy as it should do were it caused by muscular spasm. Cautley draws attention to the fact that the pylorus is normally in a state of contraction, and that dilatation occurs only in response to a stimulus. It is barely credible that the amount of spasm sufficient to oppose the stimulus to dilatation can be great enough to produce the excessive hypertrophy of the circular muscular fibers

¹ Berlin. med. Woch., 1908, No. 45.

² British Jour. Child. Dis., 1908, p. 179.

so constantly present. If the longitudinal fibers possess the power of dilating the sphincter, it cannot be explained why they do not hypertrophy as the result of pyloric spasm, for if they act as opponents of the circular muscular fibers one would expect that they would undergo hypertrophy proportionate to that of the circular sphincter. In accordance with this reasoning Cautley regards two distinct conditions as proved—a pure pylorospasm and a true hypertrophy of the circular muscular fibers, to which there may be added a spasmodic contraction from time to time.

Symptoms.—There is a striking similarity in the clinical course of all reported cases. Rarely do the symptoms begin at birth. After a period of time varying from a few days to several weeks, during which time there is no suspicion of impending trouble, the infant begins to vomit his food, at first at somewhat infrequent intervals. There may be no apparent reason for this, as the child may be breast-fed or nourished on proper scientific principles. The vomiting, however, persists and becomes more frequent, so that nearly all nourishment is immediately rejected. The infant begins to waste and looks shrivelled and marasmic. The symptoms may be now described in detail.

Time of Onset.—It is rare for the symptoms to appear before the third or fourth day of life, or after the seventh week, although the first intimation of the complaint has been recorded in the ninth week after birth. The average date of the onset is during the second to the fourth week.

Vomiting.—The vomiting begins gradually, occurring at first but once or twice a day, and having no fixed relationship to the time of feeding. Within a short time, however, the vomiting becomes more frequent, and occurs directly after the feeding, and the quantity ejected increases day by day. It may be noticed that nourishment given in small quantities may be retained, but that larger feedings are immediately ejected. As time goes on the amount required to excite vomiting grows less, so that even minute quantities of nourishment are at once returned. The vomited matters may be slightly altered from their condition when swallowed, or the milk may be curdled and admixed with mucus. Bile is almost invariably absent from the vomitus, although in two of the reported cases bile was present, causing considerable uncertainty in the diagnosis.

Characteristic of infantile stenosis is the remarkable violence of the vomiting, the vomited matter being ejected with great force, often projectile in character, or even forced through the nostrils. Equally diagnostic of the condition is the vomiting of a large quantity of food when only a small quantity has been previously given, showing that the vomit represents more than one feeding, perhaps the accumu-

lation of several that have been retained in the dilated stomach. The characteristics of the vomiting as given by Still are:

1. Its forcible character.
2. Its occurrence in a child that has been carefully fed.
3. Its persistence in spite of changes in diet.
4. A quantity returned more than that recently taken, suggesting accumulation in the stomach.
5. Its association with constipation, for usually vomiting in infants goes with loose, slimy stools.

The general condition generally goes from bad to worse, the infant loses steadily in weight, proportionate to the severity of the vomiting, often weighing toward the close of the disease barely three or four pounds. The face becomes pinched and shrunken and the child has an old and wizened look. The temperature runs a subnormal course. Constipation is well-nigh invariable, as would be naturally inferred from the impossibility of sufficient nourishment passing into the bowel to give substance to the stools. Convulsions may appear from time to time, either from desiccation of the tissues or from toxic absorption of the accumulation of food in the stomach.

Physical Signs.—Physical signs in the early days of the disease may not be apparent, but after the symptoms have existed a short time examination affords conclusive proof of the presence of the disease. Inspection shows that from time to time the outline of the stomach is distinctly visible and that faint waves of contraction pass from left to right over the surface of the organ. Reversed peristalsis may also be evident just preceding the act of vomiting. As time goes on the peristaltic waves become more and more evident, so there may be visible lumps varying in size from a walnut to a tangerine, arising at the left costal margin and passing slowly across to the right. Such a stately peristaltic wave may be followed by 2 or 3 similar elevations resembling a chain of hills. These peristaltic waves are distinctly visible two or three yards away, thus distinguishing them from the feeble peristalsis which is often observed passing over the stomachs of infants who suffer from vomiting and constipation, which can be seen only by a strong and oblique light. These peristaltic waves are easily recognized as quite different from the irregular contractions of the abdominal wall occasionally seen in squirming infants. It should be remembered that visible peristalsis occurs only at certain intervals, and therefore the examiner should take sufficient time in his search. A patient examination often occupying ten or fifteen minutes may be necessary before the signs can be elicited, and occasionally a second or even a third examination may be required. Moreover, the abdomen should be examined immediately after the infant has been fed, for the peristalsis

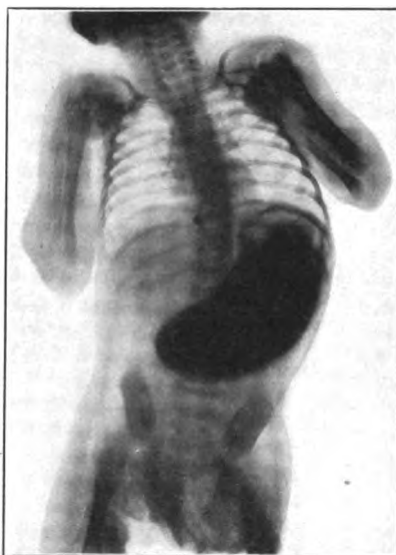
may not be evident at any other time than this. The stomach is usually quite dilated in size and somewhat inflated, causing a prominence in the epigastrium in striking contrast to the collapsed and sunken appearance of the lower part of the abdomen overlying the empty intestines.

A tumor is often palpable in the pyloric region, usually in the right nipple line, one-third of the way between the umbilicus and the right costal arch, and seems hard to the touch, cylindrical in form, and freely movable. Such a mass may not be noticed until after several weeks of vomiting, and even then may be demonstrable at certain times and not at others, for the presence of a tumor depends upon the temporary condition of the muscle at the pylorus. If the muscle be in a condition of tonic contraction a tumor is felt, but if the muscular tissue be relaxed; the pylorus may become so soft that it eludes the most experienced touch. For this reason the tumor is usually most easily detected during the period of evident peristalsis, and becomes less evident when the stomach is in a relaxed condition.

It has been recommended that the examination for the growth should be made under an anæsthetic in doubtful cases. It would seem, however, that the effect of the anæsthetic would be to relax the stomach, abolish the tonic contraction at the pylorus, and render detection most difficult. The most reasonable time for examination would be directly after the feeding, when peristalsis and tonic contraction of the pylorus are most marked.

Prognosis.—The prognosis is extremely serious, the mortality being about 50 per cent. The mortality under varied conditions of treatment, medical and surgical, is well illustrated by Still¹ in a verbal report at the Clinical Society of London. Of 23 cases under his personal observation, 14 recovered (8 after surgical treatment, 6 after medical treatment). Of the 9 that died, 3 were untreated, 3 died after operation, 3 died after medical treatment.

FIG. 75



Infantile pyloric stenosis. (Case of Dr. Charles L. Gibson; radiologist, Dr. Le Wald.)

¹ *Lancet*, March 16, 1907, p. 734.

Treatment.—Recognizing that in infantile stenosis there exists a definite obstruction to the onward passage of food into the intestine, the natural inference would be that surgical intervention would constitute the one and only form of treatment. The danger is that the brilliancy of surgical operation leads us into the error of resorting to surgery as a routine.

If the stenosis be due to an irremediable hyperplasia of the pylorus, surgery would afford the only means for relief. If, on the other hand, compensatory hypertrophy be a phenomenon secondary to pylorospasm, we have reason for attempting to relieve the condition by medical means before resorting to an operation. It is, therefore advisable to see that the medical treatment is carefully and judiciously carried out for a certain period of time at least before advising surgical interference in these young infants.

The medical treatment consists chiefly in the washing of the stomach and the regulation of the diet.

The stomach should be washed at least once a day for a prolonged period, and in bad cases lavage twice a day may be required. Plain water may be used, or a weak solution of bicarbonate of soda, 2 grains to an ounce. It is a simple process in young infants, and if properly done leads to no discomfort or disturbance.

It should be done before the feeding, at a time when under normal conditions the stomach should be empty. After lavage has been continued for a certain time there is usually a marked improvement in the infant. The vomiting ceases, the bowels begin to act normally, the visible peristalsis becomes less evident, and finally disappears, and the child improves in apparent comfort and begins to gain in weight. Improvement in the vomiting by lavage is often misleading, as the vomiting may cease within a few days after beginning the treatment even though the residual food may be as great as before, showing no actual improvement in the stenotic process. The obvious sign of improvement is a diminution in the quantity of residual food that is washed out, showing that a certain part of the food at least is making its way into the intestine. This improvement both in objective and subjective signs may appear quite unexpectedly in the very worst cases of marasmic infants with repeated vomiting and well-marked gastric peristalsis, just the sort of case which would seem hopeless at the start.

Dietetic Treatment.—The effect of change of diet is most evident when previous errors have existed. In infants who have been scientifically nourished or who are breast-fed very little can be done by changing the character of their food. The amount of nourishment taken at each feeding is, however, quite important, as small quantities

at short intervals, such as 1 or 2 ounces every one or two hours may be retained, while larger amounts of the same nourishment may be ejected.

Much information can be obtained by inspection of the residual food removed from the stomach by lavage. To pass the narrowed pylorus, the nourishment should be flocculent and semiliquid. If the milk in a given case returns curdled, a change in nourishment is indicated, either the use of whey or of peptonized milk, or any form of modified milk that is not capable of gross coagulation.

Diarrhea may come on during the treatment as a complication. The intestine not being used to the presence of chyme seems to become irritated and to give rise to diarrhea, always serious, and occasionally fatal. When such a diarrhea occurs the food should be reduced to half quantities at least, and it is claimed that half-grain doses of gray powder twice or three times a day are of service.

Drug Treatment.—Drugs of antispasmodic nature are of very little use. Bromides, ethereal spirits, valerian, and the various preparations of opium have been recommended, but the benefits are not comparable with those derived from lavage and regulation of the diet, and the use of drugs in young and weak infants is often fraught with danger.

Saline solution may be given by rectum to supply fluid to the desiccated tissues.

The length of time medical treatment should be continued is a matter of nice judgment on the part of the physician. If the patient seems to be gaining, then there is no necessity for a hasty resort to surgery, and the medical treatment may then be continued as long as the infant continues to improve. A certain degree of improvement at least is expected in nearly all cases. When this improvement comes to a standstill with symptoms of the original malady still persisting, though of a diminished severity, the time has come to decide whether the risk of operation with a prospect of complete restoration is not preferable to the prolongation of a medical treatment that ceases to be beneficial. Gastrojejunostomy or pyloroplasty should then be recommended.

CONGENITAL STENOSIS IN ADULTS

In 1879 Landerer observed the case of a man, aged forty-five years, who for years had suffered from stomach disorder. After death there was found an enormously dilated stomach without structural change or thickening at the pylorus, although the outlet was so small as to measure only 2 mm. in diameter. Landerer collected 9 other instances of narrowing of the pylorus in those between forty-three and sixty-three

years of age in his postmortem experience, although in these instances a previous history was not available.

The writer has seen one instance of this congenital smallness of the pyloric orifice.

A man, aged fifty-two years, entered the hospital with arteriosclerosis, dilatation of the heart with decompensation symptoms, and died on the fifth day after admission without having given any obvious gastric symptoms.

Autopsy revealed a large, thin-walled stomach. The pylorus was normal in every respect except that it was infantile in size, barely admitting a slate-pencil. There was no overgrowth of muscular tissue or any signs of active or healed ulceration.

Maier,¹ in 1885, contributed 31 cases found at autopsy in which the condition seemed to be congenital. The ages varied from 12 to 75, and in no instance was the narrowing associated with any gross structural change of the pylorus, although in certain of his cases an appreciable amount of thickening of the pylorus was found, apparently of congenital origin. This was before Hirschsprung's paper describing the hypertrophic congenital stenosis in infancy. On reading Maier's reports we are struck with the resemblance of certain of his cases with those of pyloric stenosis of infancy, so that it would seem that mild degrees of the infantile form not incompatible with life might persist and be evident even in extreme age.

Russell² calls attention to the fact that cases of supposed congenital narrowing of the pylorus as described by Maier are, however, not seen in infancy, so that the nature of the condition is rendered obscure.

Maylard³ reports having encountered narrowing of the pylorus which was difficult to explain on the basis of any organic or functional derangement. It was either found difficult to insert the index finger into the pyloric orifice or the finger was felt to be gripped by a uniformly narrowed ring without indication of cicatrization from ulcer or any evidence of spasmodic closure. The only construction possible, according to this writer, is that the condition represents a congenital abnormality, the aperture not developing sufficiently to meet the normal requirements. Maylard reports in his article 12 cases in addition to 7 previously reported, but of these 6 gave a history of the vomiting of blood, so that doubt is thrown upon the accuracy of his observations.

Russell⁴ reports 3 cases of supposed congenital origin, which seem to the writer to be more conclusive, although localized fibrosis of the pyloric canal cannot be ruled out.

¹ Virch. Arch., 1885, cii, 413.

³ Ibid., 1908.

² British Med. Jour., July 11, 1912.

⁴ Lancet, June 20, 1908.

It is reasonable to suppose that congenital pyloric stenosis may be encountered in adult life, either as the end result of a hypertrophic pyloric stenosis of infancy that has not been severe enough to cause the early death of the patient, or from malformations of the pyloric canal, usually of the funnel-shape described by Maier, which has persisted during the life of the individual. That instances of either form of the disorder are rare cannot be doubted.

Dr. Wollstein, of the Babies Hospital, in New York, in a verbal communication states that mild forms of pyloric stenosis due to an increase in the circular muscular fibers of the pylorus, of the congenital infantile type, have not been encountered at post mortems done on children dying of intercurrent disease. In a letter to the author Dr. Codman, of Boston, writes that at the Massachusetts General Hospital there has been no case of pyloric stenosis in adults comparable to that of infants—that is, due to muscular hypertrophy without lesion of the mucosa. His opinion is based on the operation records of the hospital from 1877 until the present time (September, 1912). Dr. Finney, on the other hand, writes from Baltimore: "In half a dozen or more cases there was a distinct thickening and hypertrophy of the circular muscle about the pylorus, without any demonstrable lesion, old or recent, in the mucous membrane. I thought at the time, and still do think, that it was probably congenital."

Dr. W. J. Mayo writes, in response to a letter of inquiry: "We have seen three or four cases of the type you mention, in which there was a history of trouble in infancy and more or less trouble up to the time of operation, in which the condition resembled pyloric stenosis of infancy, and in which great muscular thickening existed."

Symptoms.—There is a long history of stomach trouble dating back to childhood, characterized by acute attacks of greater severity. The symptoms in the main are those of chronic pyloric stenosis of the benign acquired form, distress after meals, evidences of increased peristalsis, and the vomiting of food. The gastric analyses and physical diagnosis are identical with those of the ordinary benign form, and the diagnosis can only be made if these signs and symptoms date back to childhood and if a careful history elicited no data that would suggest previous ulceration or perigastric adhesions.

Treatment.—The treatment is that of the benign form of acquired stenosis.

CHAPTER XIV

HOURL-GLASS STOMACH

HOURL-GLASS stomach is the condition in which the stomach is divided into two portions by a constriction at any point between the cardiac and the pyloric orifice. The term is a useful one in many ways, although it is hardly accurate when applied to cases in which three or more pouches exist, and to which the term segmented stomach seems to be more applicable.

Forms.—Hour-glass contraction may occur in one of three forms:

1. Congenital.
2. Acquired organic.
3. Functional.

Congenital Form.—The congenital form is extremely rare, so that its occurrence has generally been doubted. Morgagni believed strongly in heredity, and mentions a family in which three generations presented this defect. Other writers regard the anomaly as defective development, the pylorus part remaining of the intestinal type, while the cardia end alone expands to form the true stomach.

Sandifort has observed an hour-glass stomach in a fetus. Whether this case was one of defective development, or the result of intra-uterine gastric ulcer which had undergone spontaneous cure leaving the stomach deformed, cannot be proved.

Acquired Form.—Acquired organic hour-glass stomach is the ordinary form seen in operative cases. Schomorus in 1014 gastric operations found 7 per cent. presented this deformity. It is probable that with the increasing number of gastric operations during the past few years, later statistics will show the condition to be somewhat less frequent than this.

The cause for the acquired form is either ulcer, cancer, or perigastric adhesions. Of these ulcer is the most frequent cause for the deformity. Saddle-back ulcers of the lesser curvature are more frequently followed by the condition than similar lesions in other parts of the stomach. Postulcerous hour-glass stomach can be produced by the contraction and induration accompanying the healing of the ulcer, so that the lower curvature is pulled up toward the lesser to form a constriction ring, the upper portion of which is composed of dense scar tissue, the lower portion of normal stomach wall. In other instances the ulcer

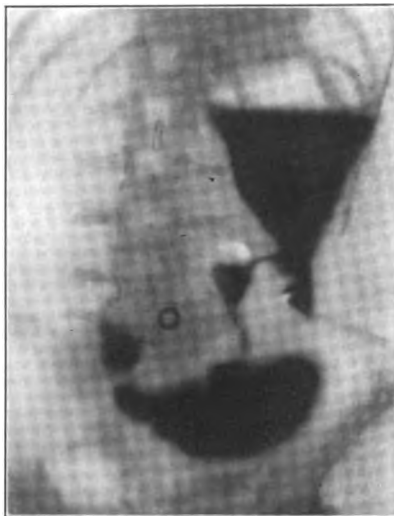
PLATE IX

Fig. 1



Tight Organic Hour-glass Stomach. The neck does not emerge from the most dependent part of the upper segment. (Radiologist, Dr. Leaming.)

Fig. 2



Trifid or Tripartite Stomach. (Case of Dr. Charles L. Gibson; radiologist, Dr. Le Wald.)

PLATE X

Fig. 1



Fig. 2



Fig. 1.—Hour-glass Stomach. Plate taken shortly after the bismuth meal, showing pouch-like sagging of the upper segment lying in front of the neck and obscuring it. (Radiologist, Dr. Leaming.)

Fig. 2.—Hour-glass Stomach. Same case as Fig. 1, taken two hours later, showing bismuth residue still remaining in the upper segment. (Radiologist, Dr. Leaming.)

Fig. 3



Spasmodic Hour-glass Stomach. Clinical history of prolonged vomiting and emaciation. Exploration showed normal stomach and was followed by a cessation of all symptoms. (Case of Dr. H. H. M. Lyle; radiologist, Dr. Le Wald.)

may be more circular, so that the constricting ring is largely composed of scar tissue alone.

In the great majority of instances, subdivision of the stomach into two pouches occurs 3 or 4 inches from the pyloric orifice. In Schomérus' series of 98 cases, 51 were near the pylorus, 34 midway, and 13 near the cardia. When near the cardia, the pyloric pouch is large, so that the cardiac portion may be overlooked. There have been instances in which anastomosis was done between the pyloric pouch and the jejunum without benefit to the patient, from lack of care in determining the point of obstruction.

In cases of multiple ulcerations hour-glass stomach may be complicated by pyloric stenosis or by similar contractions in the duodenum. Such cases have been reported by Moynihan and W. J. Mayo. Similar contraction deformities may follow corrosive poisoning or the healing of syphilitic ulcers.

Hour-glass stomach from cancer is rare except in malignancy implanted on a chronic ulcer. The mode of origin is the same as in the postulcerous form except that the lumen of the isthmus may be encroached upon by the malignant growth.

Hour-glass contraction may be due to adhesions. Connective tissue bands may pass as a bridle from one curvature to the other, drawing them together and rendering the stomach wall undistensible in the constricting line, or a band may pass from the stomach to the anterior abdominal wall in such a manner that the stomach hangs over it on each side like saddle-bags. Langerhans describes a case in which there was a cicatrix in the middle of the lesser curvature and the corresponding portion of the posterior wall, from which a peritoneal band passed to its insertion in the anterior abdominal wall, so completely contracting the body of the stomach that the finger could barely be passed through the constricted portion.

An interesting type of hour-glass stomach may occur when adhesions form between ulcers of the lesser curvature and the under surface of the liver. When the patient stands a line of tension is produced diagonally downward across the stomach holding up the greater curvature at this point, while on either side of this line the lower border of the stomach sags perceptibly downward, producing the semblance of an hour-glass stomach which disappears when the patient lies down.

Hour-glass stomach is a predisposing cause for volvulus, the pyloric portion usually rotating upward and to the left. The cardiac pouch in these cases is not involved in the torsion so that the cardiac orifice remains patent and the swallowing of food and vomiting are possible.

Symptoms.—The symptoms at first depend largely upon the cause, be it ulcer, cancer, or perigastritis, but when the deformity has fully developed the symptoms are more distinctive and resemble those of stenosis either in the pyloric or the cardiac orifice. If the constriction ring be near the cardia the symptoms are those of difficulty in swallowing and the immediate regurgitation or vomiting of recently ingested food, so that from the history alone it may be difficult to decide between an esophageal diverticulum and an hour-glass contraction at the cardiac end of the stomach. If the contraction be in the middle or toward the pyloric end the symptoms are those of pyloric stenosis. Until the

FIG. 76



Spasmodic hour-glass stomach. Incisure of greater curvature from ulcer of lesser curvature adherent to the liver. Sketch made at the time of operation. (From Dr. J. C. Bloodgood.)

adoption of the *x*-ray in diagnosis the condition was usually found at autopsy or quite unexpectedly at operation, no suspicion of its existence having been entertained but by the adoption of this modern means of examination, the diagnosis is being made in an increasing number of cases. This is shown by the fact that Moynihan diagnosed the condition correctly in one out of six of his first series, and in seven out of nine of the cases that came later under his observation.

Physical Signs.—The diagnosis of hour-glass contraction by physical signs is made either by the use of a tube or by the *x*-ray.

Examination by Means of the Tube.—A number of tests have been recorded for the detection of the deformity, which are more or less

valuable, but are at the present time so inferior in accuracy to a radiographic examination that they are not regarded as bearing more than corroborative testimony to the existence of the disorder. Their greater value consists in arousing suspicion of the disorder when they occur in the routine examination of a patient with serious indigestion, or under conditions in which facilities for an x-ray examination are not at hand.

The following tests may be enumerated:

1. By the induction of water through the tube there may be a visible prominence in the left hypochondrium, subsiding in a few seconds and then appearing further over to the right side. The passage of the liquid through the isthmus is usually accompanied by audible gurgling sounds:

2. When water is so introduced, it may be found that the larger part cannot be removed from the stomach by aspiration, suggesting that it has passed into the second pouch. This is known as "Wolfer's first sign." The writer regards it as absolutely inconclusive, as it is often difficult to recover water from an atonic stomach that is otherwise normal.

3. If water be introduced that cannot be recovered, even though splashings be heard indicating the pressure of liquid in the stomach, it may be that the fluid has flowed into the pyloric pouch, where it cannot be reached by the tube. This is known as the "paradoxical dilatation" of Jaworski. It is not a sign of much value.

4. If on washing the stomach the water returns for a time quite clear and then suddenly there occurs a gush of cloudy fluid, it would indicate the passage of gastric contents from a filled pyloric pouch into the cardiac sacculatation that has been cleansed by the lavage. This is known as "Wolfer's second sign," and is regarded by Mayo Robson as possessing considerable diagnostic significance. When this phenomenon occurs, suspicion of hour-glass stomach should always be entertained, but the writer has found this sign present when no hour-glass contraction has existed.

5. Inflation of the stomach by artificial dilatation may show a distention of the upper pouch which subsides as the gas passes through the isthmus to dilate the stomach more generally. The outline of the biloculated stomach may then be apparent.

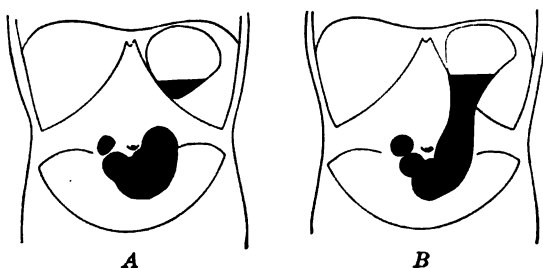
6. The transillumination of the cardiac pouch by gastroduaphany is totally inaccurate and unsatisfactory.

Radiographic Diagnosis.—Organic Hour-glass.—The organ is seen to consist of two sacs connected by a narrow isthmus, the appearance being the same in all the plates. Very characteristic is it when both sacs contain bismuth surmounted by an air-bubble. Equally character-

istic is the fact that the neck does not emerge from the most dependent portion of the upper sac, but from a point higher up, the lower portion of the upper segment passing downward and to the left from the emergence of the communicating channel. The upper segment is usually more dense in shadow than the lower one, and repeated examinations, as by the fluoroscopic method, show that the upper sac empties itself gradually into the lower cavity.

The isthmus is usually smooth in ulcer, shows nodular indentations if cancerous stricture be present. Should the isthmus pass from the posterior surface of the upper sac, the channel may be obscured by the most dependent portion of the upper cavity hanging like a curtain before it. The six-hour plate may show a bowl-shaped food residue in the upper sac, still obscuring the outlines of the isthmus. These appearances are well seen in the accompanying radiographs.

FIG. 77



Spasmotic hour-glass stomach with no ulceration. *A*, spasm present; *B*, spasm disappeared after vigorous contraction of abdominal muscles. (Hertz.)

Spastic Hour-glass (Hypertonic Type).—1. In organic pyloric stenosis with increased peristalsis a contraction wave may start near the fundus and completely separate the contents of the pylorus from the rest of the stomach. Radiographs will show an apparent hour-glass, differing from the organic form in three particulars: (*a*) The neck comes from the most dependent portion of the upper segment; (*b*) the constriction ring is equally shown on upper and lower curvature; and most important of all is that (*c*) the condition appears on some plates and not on others, showing the contraction wave to be but a transient one.

2. With ulcer of the lesser curvature, a deep contraction, indenture, or incisure may be seen on the lower curvature. This appearance usually indicates adhesions to the liver, and is then apparent only when the patient stands, the line of traction often disappearing if the plates are taken in the recumbent position. In other cases a more permanent incisure is seen with lesser curvature ulcers even in the absence of adhesions, and remains fixed in a series of plates regardless

of the position of the patient. The same appearance may also be due to carcinoma of the lesser curvature, especially if engrafted on an old ulcer or adherent to the liver. According to Hertz, spasmodic hour-glass contraction may often be made to disappear by abdominal massage, contraction of the abdominal muscles, and by injections of atropine.

Before hypertonic functional hour-glass contraction can be diagnosed and the organic form excluded, a series of plates should be taken on different days, both in the erect and recumbent position. Suspicion of functional hour-glass should always be entertained whenever one deep incisure only is seen on the greater curvature, and the probability considered of the presence of an ulcer with or without adhesions on the lesser curvature. In hypertonic contraction the spasm does not seem to prevent the rapid filling of the distal pouch, nor does it lead to increased peristalsis in the proximal pouch during the continuance of the spasm (Hertz).

In spite of all precautions mistakes are made. The accompanying plate (Plate X, Fig. 3) was taken of a patient who for years had suffered from persistent epigastric pain and vomiting. The incisure was present in a fixed spot in all the plates, and food-stasis seemed to exist as bismuth was present in the stomach thirty-five hours after the bismuth meal. Exploration showed a perfectly normal stomach, free from ulcer or adhesions. A radiographic plate taken two weeks after the operation showed no evidence of the former indentation. The gastric symptoms ceased after the exploration and have never returned.

3. In other instances a portion of the lower curvature may be caught up by adhesions attached directly to the affected area. Such incisures are apt to vary materially with changes in the position of the patient, and usually disappear entirely in the Trendelenburg position.

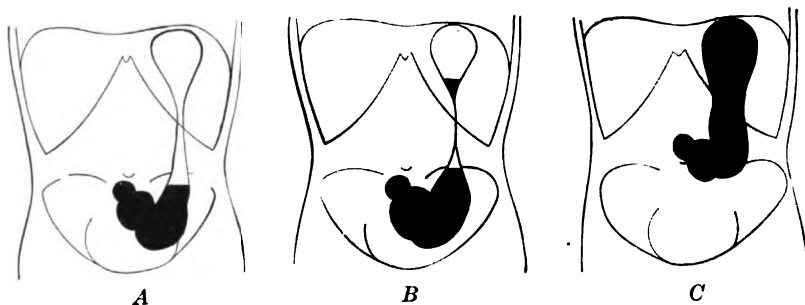
Hypotonic Hour-glass Stomach.—Hypotonic hour-glass is but rarely observed, although it is described by Hertz as not infrequent.

In combination of ptosis with extreme degrees of atony, the most dependent portions of the stomach sag more and more deeply as food is taken, and the tension exerted on the body of the stomach results in the narrowing of the passage between the fundus and the portion of the stomach that is sagged down by the weight of the food, until the lumen is finally obliterated and the stomach is divided into two segments, the upper containing the air-bubble and the lower sagged portion containing the food. As more food is taken, part of it may remain in the upper portion, so that each portion may contain food and an air-bubble. This apparent hour-glass condition disappears when the patient lies down.

It is important to remember that if the plate be taken while the

patient is on the back, the stomach may lie across the vertebral column so that the pars media is raised while both extremities of the organ sag backward, forming bismuth pools on either side, while the central portion, overlying the vertebræ, is unfilled and drawn into a somewhat narrow isthmus. The rugæ are, however, quite distinct and the apparent hour-glass disappears when the patient stands. The appearance may closely resemble tumor of the pars media.

FIG. 78



Orthostatic or hypotonic hour-glass stomach. A, vertical position, first stage; B, vertical position, second stage; C, horizontal position. (Hertz.)

Treatment.—If the isthmus between the two sacculations is of fair size, the patient may get along fairly well on the medical treatment that is adopted in cases of pyloric stenosis. By medical means alone the nutrition may be improved and the patient rendered more comfortable, but no permanent results are to be expected.

The curative treatment is entirely surgical. A number of different operations for the relief of this condition have been devised. They consist of digital divulsion; of gastroplasty analogous to the pyloroplasty of Finney; of gastrogastrostomy or the lateral anastomosis of the two pouches. Gastrojejunostomy is applicable to the cases in which the constriction occurs close to the pylorus. In some instances gastrectomy may be deemed advisable. The exact surgical technique to be adopted cannot be decided upon until the abdomen is opened.

CHAPTER XV

DIAPHRAGMATIC HERNIA—EVENTRATION—VOLVULUS

DIAPHRAGMATIC HERNIA

DIAPHRAGMATIC hernia consists in the protrusion of one or more of the abdominal viscera into the pleural cavity through a congenital defect, through a rent or tear as the result of traumatism, or through one of the natural orifices.

Forms.—According to the variety of the opening in the diaphragm, is based the division of diaphragmatic hernia into congenital or traumatic and of acquired cases.

Congenital Hernia.—Congenital hernia is the most frequent form. Of 433 cases collected by Grosser in 1899 a congenital origin could be demonstrated in 232. In Knaggs' series of 63 cases, 24 were congenital, 21 traumatic, and 8 were acquired, the herniated parts passing through one of the normal existing orifices of the diaphragm.

If it be considered that a peritoneal sac is essential to hernia, few of the reported cases, now over 600 in number, can be considered instances of hernia, as a hernial sac properly speaking does not occur with the traumatic and but rarely with the congenital form. The term is, however, used in a broad sense to include all the cases of displacement of the abdominal viscera into the thoracic cavity irrespective of whether or not they may be inclosed within a peritoneal sac. In Grosser's series of 433 cases a true sac was found in but 10 of the acquired and in 30 of the congenital cases. In 266 cases collected by Lacher a true sac was present in only 28 instances.

A distinction must be made between diaphragmatic hernia and eventration. In the latter condition there exists weakening of the dome of the diaphragm, so that under normal intra-abdominal pressure the diaphragm bulges upward to form a sacculation into which enter one or more of the abdominal viscera. The diaphragm, even though thinned and undeveloped, still remains as a limiting membrane lying between the diaphragmatic layers of the peritoneum and of the pleura so as to form a true and unbroken sac.

Diaphragmatic hernia is almost always left-sided. In Arnheim's series of 284 cases, reported by Chadbourne,¹ the right side was affected

¹ Amer. Jour. Med. Sci., 1903, 312, cxx.

in but 8 per cent., the reason for this relative immunity being obviously due to the presence of the liver which acts as a buffer, protects the diaphragm on that side from the effects of increased abdominal pressure, and closes very efficiently any defect that may occur. Almost every abdominal organ except the rectum and the pelvic viscera have been found in the hernia, as may be shown by the following table of hernial contents compiled by Rochard and Lacher:

	Rochard (330 cases).	Lacher (276 cases).
Stomach	187 times	161 times
Colon	177 times	145 times
Small intestine	133 times	83 times
Omentum	107 times	96 times
Spleen	78 times	
Liver	60 times	43 times
Duodenum	48 times	35 times
Cecum	35 times	20 times
Pancreas	32 times	27 times
Kidneys	3 times	2 times

To show how varied the contents of the hernia may be, Knaggs' complication of 59 cases will be interesting.

TABLE SHOWING THE VARIOUS ASSOCIATED VISCERA IN THE HERNIAE

Stomach alone	9
Stomach and part of duodenum	3
Stomach and omentum	5
Stomach and spleen	3
Stomach, spleen, pancreas, and colon	6
Stomach, spleen, and transverse colon	22
Stomach, liver, spleen, omentum, and small intestine	2
Stomach, liver, and small intestine	3
Stomach and almost all the abdominal viscera	1

It is seen that in Knaggs' series in which the stomach was involved, the colon was implicated in 33, the spleen in 16, and the pancreas in 5, in each instance being associated with hernia of the spleen and colon. In some cases the colon alone was involved.

Right-sided hernia are usually small and often contain small knob-like protrusions of the liver.

MECHANISM.—The mechanism of diaphragmatic hernia is frequently quite involved, so that many of the cases are difficult to understand even at autopsy. Passage of the stomach through the hernial ring is seldom a simple upward movement, but is complicated in the great majority of instances by torsion either in the longitudinal or the vertical axis, causing a complicating volvulus, often with resulting strangulation

symptoms or even perforation. When the stomach and colon are found in the hernia the colon often lies uppermost and the stomach is so twisted on its longitudinal axis that the lower curvature points upward and forward, so that the organ is upside down, seeming to prove that the colon first enters the hernial orifice and then drags the stomach after it, so that the lower curvature is the next to engage. The resulting volvulus is of the "receding car-wheel" type. This is the view taken by Payer, and it seems to be corroborated by pathological findings, although Knaggs claims that usually the stomach enters first, dragging the colon after it by means of the gastrocolic omentum. Whenever the pyloric portion of the stomach enters the esophageal or other neighboring openings of the diaphragm, as in the acquired form, a rotation on a vertical axis is bound to occur. If the opening be to the left of the cardia the rotation naturally is of 180° and the lesser omentum is sharply twisted on itself, so that the cardiac portion of the stomach that is not in the hernial sac is poorly supplied with blood, softens, and may become gangrenous. The pyloric portion, provided that the diaphragmatic opening be not too narrow, is well supplied by blood by the right coronary and gastroduodenal arteries. In the cases of Knaggs and Willetts,¹ in which a gastric hernia took place through the normal esophageal opening (Knaggs' to the left of the cardia, Willett's to the right) gangrene of the cardia occurred, while the herniated portion was easily reducible and of good nutrition.

If the hernial opening be of small size, the portions engaging in the orifice may be compressed by tumefaction or by the wedging in of a portion of the omentum, so that strangulation of the portions of the viscera within the thoracic cavity may occur, rarely, however, at the seat of actual constriction. Gangrene may also result or splits through the peritoneal, or muscular coats produced by extreme distention may determine the site of perforation whenever the softening mucous membrane gives way. The pleura may participate in the inflammation and a considerable pleuritic exudate may result. When the aperture is small, either the esophagus may be compressed or twisted, so that deglutition is impossible, or should similar obstruction occur at the duodenal end nothing can pass downward. The herniated portions may for considerable periods of time be free to enter the hernial orifice, and after a time undergo spontaneous reduction. This is the history of many patients whose hernial orifice is large and free from adhesions. When adhesions occur in the vicinity of the aperture, spontaneous reduction may be rendered impossible, and the condition becomes more or less permanent. Distention of the portion of the stomach

¹ *Lancet*, August 6, 1904

included in the hernia almost invariably occurs, either as a transient condition capable of natural relief, or permanent and progressive, especially if strangulation or torsion should occur.

Congenital Diaphragmatic Hernia.—Congenital diaphragmatic hernia takes place either through some developmental defect of the diaphragm or through unclosed pleuroperitoneal passages, the latter being the route taken in 21 of 26 cases reported by Keith.¹

Hernia through the left pleuroperitoneal passage is the usual route in the congenital cases, although defects may be at any portion of the diaphragm or may even take place through the normal esophageal opening. Defects in the right leaflet are apt to be blocked by protrusions of the liver. The aperture may vary in size from 2 cm. in diameter to the entire absence of half the diaphragm. Extensive congenital defects of the diaphragm are frequently accompanied by anomalous lack of development of the lung. In a case reported by Beckman² exploratory operation revealed congenital absence of the diaphragm and lung on the left side.

Traumatic Hernia.—Rents and tears from trauma may occur in a greater variety of situations than in the congenital form, although the closure of the aperture by the liver in case of right-sided lacerations usually prevents visceral intrusion. Laceration of the diaphragm may be due to stab wounds or to bullet injuries, to crushing of the body, as in buffer or coupling accidents, or being run over, to severe blows upon the abdomen or lower thorax, or to falls from a height. A sudden doubling up of the body with the chest between the knees, as in sand or gravel slides, may be the cause for the tearing of the diaphragm tissue. In 21 traumatic cases collected by Knaggs, 7 were due to bullet or stab wounds, 8 to crushes, 6 to falls from a height.

The orifice at first is of the nature of a rent or tear, but if the patient survives it becomes more or less circular with callous well-defined edges which may be adherent to neighboring parts or to such viscera as may be found passing through it. The injury may be a serious one, as in stab wounds or body crushing, or the traumatism may be apparently insignificant and not attended by any symptoms that are apparently serious at the time. In a case reported by Howe,³ a young man on a bicycle ran into a rubbish cart, receiving a blow from the shaft just below the left breast. The injury was apparently so slight that in six days the effects of the contusion had passed and he was able to reassume his life at a military school. Symptoms of strangulation occurred

¹ British Med. Jour., 1910, ii, 1297.

² Surg., Gynec., and Obstet., August, 1909, p. 154.

³ Medical News, November, 1901, p. 845.

seven months later, after a jumping contest. Autopsy showed the stomach, transverse and upper part of the descending colon, with the greater part of the omentum within the pleural cavity. The omentum was adherent to the pleura at the site of the contusion of the chest.

Acquired Form.—In the acquired form the intrusion takes place through one of the natural openings of the diaphragm and constitutes a true hernia in the sense that a hernial sac regularly forms an investing membrane. Hernia through the esophageal opening is most common, either to the right or the left side, and may be of considerable size. In the case reported by Beckman¹ one could introduce the hand at the site of the esophagus. Next in frequency is hernia through the opening for the splanchnic nerves and for the aorta.

Symptoms.—Congenital Form.—In the congenital cases the symptoms may appear at or soon after birth or may be deferred until late in life.

Lacher estimates that 40 per cent. of all the congenital forms of hernia give symptoms during an extremely early age. Knaggs in 24 cases of this form of hernia found that 4 occurred in the fetus, 5 in children who died at birth, while in 15 the symptoms first appeared between the ages of six weeks and sixty years.

In many instances the child is stillborn or dies soon after birth. In the latter instance cyanosis and dyspnea are prominent symptoms, and the left chest does not usually expand to a normal degree. Dextrocardia is usually present, and death results within a few hours.

In other instances the child may survive days or even weeks in a condition of poor nutrition and with impaired respiratory powers. Gastric symptoms may be entirely lacking. These children frequently die from intercurrent disease, especially of a pulmonary nature.

It may happen that no symptoms are evident until late in life. In these cases of late development of symptoms, there is a congenital defect in the diaphragm through which the stomach and omentum are apt to pass. No symptoms are usually induced by such a condition until the time arrives when the herniated portions undergo strangulation, either from volvulus or twist, or from the engagement of other abdominal organs into the hernial cleft or to a swelling of the herniated portions themselves by accumulation of gas within them.

Ringrose² reports the case of a woman, aged twenty-six years, who had never suffered from indigestive troubles until three days before her death, when in the seventh month of pregnancy she began to vomit yellow fluid and complain of pain in the upper left abdomen. The following day she miscarried without labor pains or warning; she became

¹ Loc. cit.

² British Med. Jour., November 26, 1910, p. 1673.

cyanotic, vomited blackish fluid, had intestinal pain in the left side under the costal arch, developed jaundice, and died after an illness of three days. A distended stomach, containing 3 quarts of blackish fluid, was found in the left chest, reaching as high as the second rib. The pylorus was found in its normal position, but there was an hour-glass constriction where a portion of the stomach had passed through the hernial cleft in the diaphragm and had become strangulated. The whole stomach was rotated on its longitudinal axis so as to lie upside down. One-quarter of the small intestine, and a greater part of the omentum, had passed through the opening and had compressed the left lung against the vertebral column. The hernial orifice in the left side of the diaphragm admitted the entire hand, and was unmistakably of congenital origin.

In other cases there may be attacks of mild and transient strangulation, the patient complains of mild indigestion after eating, and from time to time of epigastric pain and efforts at vomiting, although at any time, overdistention of the herniated stomach may cause sudden heart failure and even death. Recovery follows the spontaneous reduction of the hernia, but the attacks tend to become more frequent and more severe until the time arrives at which the herniated portion becomes entirely strangulated.

Fisher¹ reports the case of a man, who for years would have attacks of pain after food, so severe that he would roll on the floor until the pain was relieved by vomiting. For two months prior to his last illness the attacks had been growing more severe and more frequent. October 17, at 5 P.M., one of these attacks began with epigastric pain and distention. The following day he vomited brown fluid and his pain was more severe. The distention in the epigastrium had likewise increased, and a swelling could be made out in the left hypochondrium, feeling like an inflated rubber bag. There was tympany in the anterior axillary line as high as the sixth rib. He died at 10 P.M., October 19, after an illness of thirty hours' duration.

The autopsy showed free gas in the abdominal cavity. The lesser peritoneal cavity was distended and filled with thick black fluid, looking like altered blood. There was found an aperture in the diaphragm, two inches in diameter, to the left of the esophageal opening, apparently of congenital origin, and the stomach had been in the habit of passing freely into the hernial sac, from time to time; and then after vomiting, undergoing spontaneous reduction. Through this opening the pyloric portion of the stomach had become herniated though easily reduced by traction. The cardiac portion, lying in the abdominal cavity, had

¹ *Lancet*, December 8, 1897, p. 1584.

perforated into the lesser peritoneal sac, and through the perforation dark red fluid was oozing.

Traumatic Form.—In the traumatic cases the onset of symptoms may directly follow the accident or may be deferred until months or years afterward. According to Lacher, of 36 cases of injury to the diaphragm that were not operated upon immediately, 5 died in one day, 10 in a month, 5 in five years, and 5 in twenty years.

The initial symptoms are those of the causal injury, usually with considerable degree of shock. In the case of stab wounds or bullet injury pneumothorax may result and there may be symptoms of internal hemorrhage and the patient may die from the injury before gastric symptoms have sufficient time to develop. The diaphragmatic wound rarely in itself is the cause for death. Should the patient survive the initial shock of the accident, distressing dyspnea and severe epigastric or thoracic pain are the chief symptoms observed. The face is usually somewhat cyanotic, the breathing shallow and difficult. Vomiting may occur, or there may be ineffectual attempts to vomit and a desire but not an ability to raise wind. The symptoms of the acute onset are strikingly like those of sudden pneumothorax. Strangulation or perforation may supervene.

In the majority of instances a gradual improvement takes place and the patient is thought to have made a satisfactory recovery, although there may remain some epigastric pain or a moderate degree of dyspnea. In other instances there may be no indication of any digestive trouble whatever until the appearance of attacks which mark the temporary incarceration of the herniated organs.

Pain is the chief symptom and is often so excruciating that the patient will roll on the floor in agony or will scream so that he may be heard for blocks. Dyspnea usually results from the pressure of the herniated viscera on the heart or lung, and is regularly more marked when the hernia is left-sided. During the periods of most intense dyspnea cyanosis is usually present. Dyspnea may continue after the acuteness of the attack has subsided, so that it becomes more or less permanent and may last for years, appearing after exercise or after the taking of a full meal.

Vomiting is usually present during the acute exacerbations, the vomited matters often consisting of a brownish blood-stained fluid. Hemorrhage from the stomach is almost invariably present in severe attacks. In cases of torsion or compression of the esophagus vomiting is impossible, though futile attempts to empty the stomach by repeated and painful retching are not uncommon. Thirst in all cases is excessive and is not easily assuaged.

When the hernia is chronic there is often the history of recurring pain and vomiting, especially after meals, these symptoms probably

being induced by obstruction to the passage of food from the herniated stomach into the intestine. Some patients complain of pain regularly occurring when they rise in the morning, which disappears after they have been up and around for a time. An explanation of this important symptom is that while lying down the contents of the abdomen gradually find their way into the chest. The pain on rising is caused by the crowding of the bowel into the opening by gravitation, and the relief which appears after a time seems due to the return of the contents to their proper position in the abdomen. The full feeling of extreme distention of the stomach from gas is often the source for complaint in the chronic cases. Gastric tetany has been known to occur.

Should strangulation occur, the symptoms become suddenly intensified and collapse supervenes. There may be inability to swallow liquids, or there may be watery blackish vomiting, according to the patency of the esophagus. Symptoms of acute perforation may occur. Death usually takes place within forty-eight hours after the onset of strangulation symptoms.

Acquired Form.—The symptoms in this form are apt to appear in a series of minor attacks without apparent cause, terminating the sudden onset of strangulation symptoms unless relieved by timely intervention.

In a case reported by Beckman¹ which was operated upon successfully by W. J. Mayo, the clinical symptoms and gastric analysis closely resembled carcinoma, although from the physical examination a cystic gall-bladder was diagnosticated. The case is reported as follows:

Woman, aged forty-seven years, who four years previously began to suffer from sharp severe pain in the pit of the stomach and to the left side one-half hour after meals. She would vomit if she ate more than a moderate quantity of food at a single meal. Occasionally she vomited blood, and food eaten two or three days previously. At night she might vomit the food she ate at noon. These symptoms have grown much worse the past year, so that she is unable to keep food on her stomach more than an hour, has lost 100 pounds in the past three years, and is much emaciated.

On examination a small freely movable tumor, the size of a lemon, is palpable under the right costal arch, which on the exploration proved to be a cystic gall-bladder.

Gastric analysis showed total acidity of 5, no free hydrochloric acid.

At operation by W. J. Mayo it was found that the esophageal opening would admit the hand, and that through it had passed the entire stomach, quite adherent inside the thoracic cavity. The adhesions

¹ Loc. cit.

were tied off and the stomach was sutured, the dome to the margins of the diaphragmatic opening, the body at various points to the parietal peritoneum, and the pylorus and duodenum were drawn to the right side and held in place by sutures. Recovery uneventful.

In some instances the attacks of temporary incarceration are attended by symptoms so slight as to be almost passed over in the clinical history, so that the diagnosis is made with greater difficulty than usual. Waller¹ reports the case of a young man, aged twenty years, whom on arrival he found dead. From the family the following history was obtained:

Five years previously a heavy farm cart passed over his left chest and he was obliged to remain in the hospital for five weeks in consequence. At that time he ran a slight evening temperature for a few days, although no definite lesion could be discovered. Gastric symptoms were not recorded in the history. One year after his discharge he passed an examination into a Forester's Lodge, so that apparently the chest signs were not prominent at that time. He remained well until four months before his death, when he had a "sick attack," which passed off in twenty-four hours without medical assistance. The morning before his death he was working in the fields as usual and ate his accustomed dinner. In the afternoon he began to feel ill and vomited. His condition was not considered serious enough to call a physician. The following morning he died after an illness of about eighteen hours' duration.

At autopsy the left lung was compressed to the size of an average spleen. The whole of the stomach except the pyloric end had passed through the esophageal opening of the diaphragm, the aperture admitting three fingers, was quite unconstricted, but contained a large amount of blackish fluid.

It is doubtful whether this case should be classed among the traumatic or the acquired hernias.

Diagnosis.—Physical Signs.—The physical signs should be sufficiently evident to occasion at least a suspicion of the disorder, but in the great majority of instances the physician is unprepared for the emergency and does not think of diaphragmatic hernia even though the physical signs taken in connection with the clinical history should be quite convincing. Of 250 cases reported by Liechtenstein a correct diagnosis was made in but five. These cases were, however, collected before the days of *x*-rays.

The chest on the affected side is usually prominent and comparatively motionless, the restriction in its expansion being more noticeable in the lower portion. Litten's phenomenon is regularly absent. Intestinal

¹ *Lancet*, October 15, 1910, p 1135.

peristalsis communicating writhing movements to the thoracic wall were observed by Holt in the case of an infant. Retraction of the epigastrium may be detected and there may be a considerable rigidity of the upper abdominal wall. In other cases the epigastrium may be unduly prominent, from the distention of the cardiac end of the stomach that remains on the abdominal side of the hernial orifice, and palpation may even detect this distended portion of the viscus as a smooth elastic tumor not unlike an inflated rubber bag. Scars from old wounds of the lower thorax are of much value as corroborative evidence.

The signs at the base of the chest closely resemble those due to pneumothorax. The note is tympanitic over the lower section, or there may be an area of dullness due to fluid in the herniated stomach or to the presence of spleen or omentum below the area of tympany. The note is more clearly tympanic or even amphoric after artificial inflation of the stomach by CO_2 and the area over which the note is elicited becomes greater. Filling the stomach with water may cause partial flatness instead of tympany. If the colon be part of the hernia its inflation will likewise affect the tympany in the chest. Over the affected area breath sounds are distant, and loud gurgling and tinkling sounds may be heard. In some instances these adventitious sounds are evidently synchronous with peristalsis rather than with respiration, and are therefore very suggestive of the condition, although some uncertainty may arise whether the sounds arise in the thorax or are merely "transferred" from the abdominal cavity. Similar gurgles may, however, be heard during expiration and are due to the forcing of gas through the neck of the herniated organ, whether colon or stomach, into the portion of the organ that lies below the diaphragm. The physical signs are often altered remarkably by changes in the position of the patient.

Above the affected area the note may be hyperresonant or of a dull tympanitic quality, while the breath sounds are but slightly altered from the normal. These are the signs observed in cases in which visceral encroachment on the lung has not been sufficient to cause it to be compressed. When actual compression of the lung takes place, the note becomes progressively dull and the breathing approaches the bronchial type. As the majority of hernias are left-sided the heart is usually displaced to the right. Dextrocardia is more easily demonstrated by x -rays than by percussion, as the percussion signs of the left border are apt to be obscured by the neighboring tympany.

Convincing evidence of the stomach or colon lying in the thoracic cavity is afforded by the x -ray, both before and after the filling of the stomach or colon by bismuth suspensions. The simplest method is perhaps the demonstration that the colon after bismuth injection

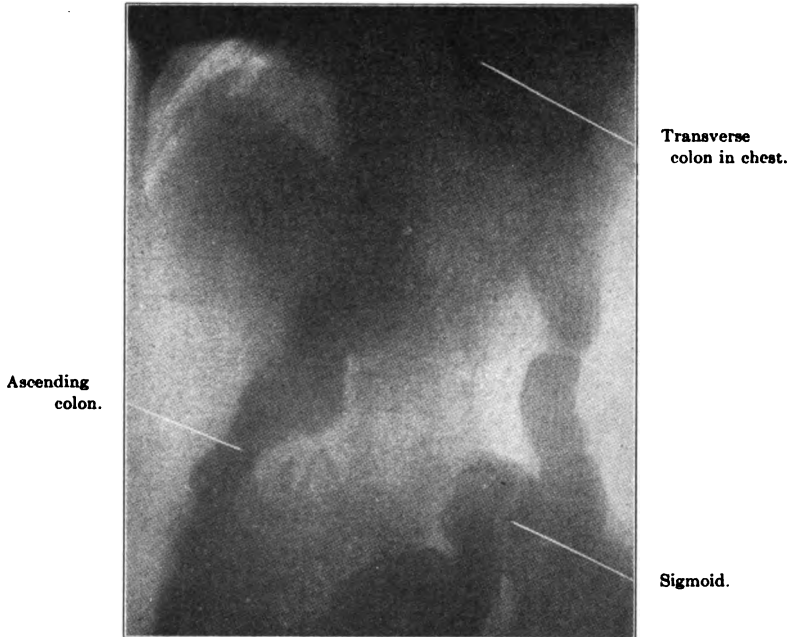
PLATE XI



Right-sided Diaphragmatic Hernia. Colon in thoracic cavity, with transposition of liver only. (Case of Dr. Rowland G. Freeman; radiologist, Dr. Leaming.)

lies in the thoracic cavity, although it is not in every case of hernia that the colon participates in the process, but a plate showing bismuth in the colon above the diaphragm line is conclusive evidence of a diaphragmatic hernia.

FIG. 79



Diaphragmatic hernia. Photograph of abdomen after an enema of bismuth had been given. (Carson-Huelsmann.)

According to Giffin the most noticeable abnormality is the existence of a curved shadow line in the left chest with the concavity downward. This shadow line generally maintains a typical dome shape whether it be high or low. The mottled appearance of lung tissue is visible through the gas contained in the stomach, a point of value, according to Giffin, in differentiating between diaphragmatic hernia and eventration in which this appearance is not noted even with extreme distention of the stomach.

A series of x-ray plates following a bismuth meal should afford convincing proof of hernia, if the stomach be identified as lying in a high position above the diaphragm. The upper level of the bismuth meal may be distinctly seen, bounded above by a large air-bubble, the upper limit of which is marked by the curved bowline which represents the upper wall of the stomach. These important points are well illustrated by the study of the accompanying plates from Giffin, which merit careful study.

A "paradoxical expiratory displacement" has been observed in diaphragmatic hernia. During forced inspiration the diaphragm descends normally on the right side while the line on the left side ascends. During forced expiration reversed movements occur. This phenomenon is said to be absent in eventration. By powerful expiratory movements of the abdominal wall the shadow line on the left side is forced high into the chest.

Differential Diagnosis.—A differential diagnosis between hernia and eventration is of importance, because while hernia is often operable, eventration is beyond the power of surgery to repair, and unless a diagnosis is made the patient may be subjected to an exploration that is futile and unnecessary. Giffin attaches much importance to the mottled lung tissue appearing through the gas-bubble in cases of hernia and to the relative position of two curved lines on the radiographic plate. "If two curved shadows be present, a radiographic or fluoroscopic examination after distention of the stomach should indicate which line is stomach and which diaphragm; if the lower line be stomach it will move up against the diaphragm line, and the pyloric end will then unfold; if the upper line be stomach, distention upward into the chest will be almost unlimited. If the bowlines shadow represent both diaphragm and stomach, distention will cause merely the above-mentioned unfolding of the pars pylorica."

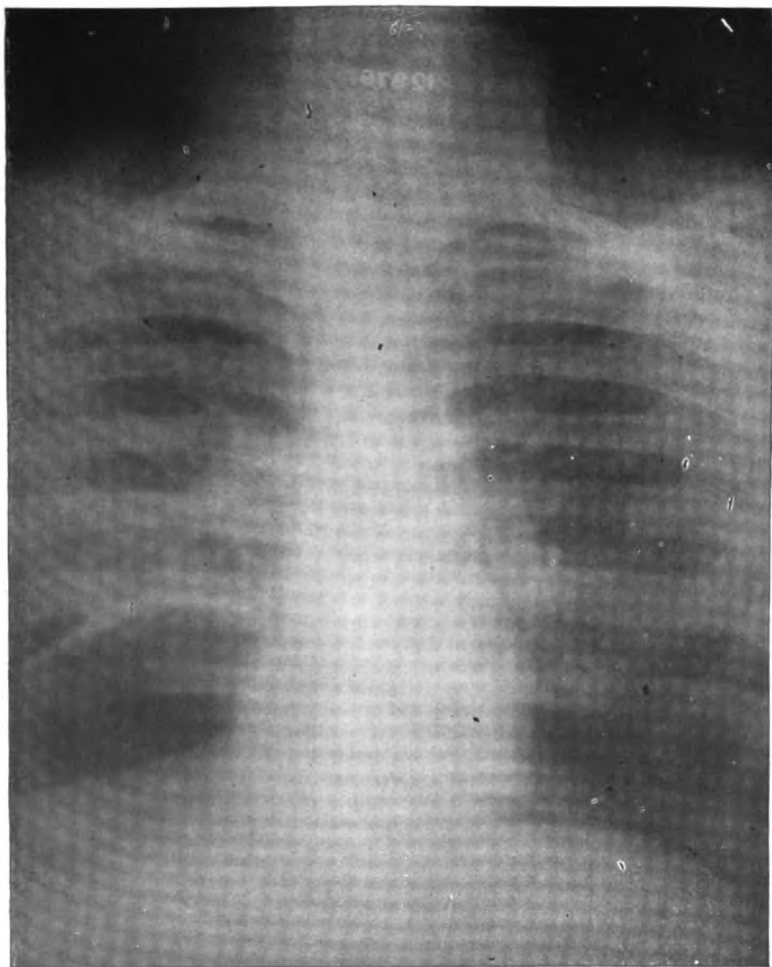
The history of recurring attacks of pain and vomiting after injuries to the lower thorax or abdomen would point to hernia rather than to eventration, but in spite of every possible care a differential diagnosis may be at times quite impossible.

In pneumothorax the physical signs of extensive involvement of the lung are usually evident above the area of tympany, and in many cases signs of pulmonary disease are obtained over the opposite side. Gastric symptoms are less in evidence, nor is there usually the history of recurring attacks of temporary incarceration so commonly elicited in the hernia cases. Radiograms in pneumothorax usually show an unbroken diaphragmatic line, whereas in hernia this line is often irregular or incomplete. The differential diagnosis while simple enough in many instances may be extremely difficult, especially when the patient has received stab or bullet wounds in the chest. In one case needling of the chest resulted in the aspiration of brandy and water that had recently been given to the patient.

Large basic cavities would hardly be mistaken for hernia if sufficient attention be given to the history and the physical signs. Radiographs would show extensive lung involvement surrounding the air space and an unbroken diaphragm line would lie below the lesion.

Subphrenic pyopneumothorax usually follows perforation of a gastric

PLATE XII



June 26, 1911. Case, diaphragmatic hernia. with the stereoscopic radiographs made of patient in the vertical position, the sternum next to the x-ray plate, care being taken to avoid any rotation of the spine on its long axis. The central focus corresponds to the level of the seventh dorsal vertebra.

We are viewing the thorax through from behind, therefore the right side of the print represents the right side of the thorax. These prints are fac-simile reproductions of the original plates.

Note the position and outline of the pericardial shadow. The heart inclines strongly toward the right, its right border extending a greater distance to the right of the midsternal line than does the left border to the left of the spine.

Both hiluses are abnormally dense, the left being markedly enlarged. Both apices are clear, and the pulmonary tissue is normal in all three right lobes and in the upper left lobe.

In the lower portion of the left thorax can be seen clearly a curved white line of greatly increased density. The proximal end of this line is opposite the level of the spinal juncture of the eighth rib on the left side. From this point the line curves outward along the eighth interspace to the ninth rib. Between this line and the upper border of the tenth rib is seen a dark area of greatly decreased density, through which one can clearly distinguish branches from the lower pole of the left hilus. The costal portions of three ribs are also visible. The left margin of the pericardial shadow is not clearly defined.

Note the clear-cut outline of the dome of the diaphragm to the right of the spine and compare it with the faint, indistinct outline on the left of the spine, which is to be seen just below the tenth rib.

The unusual appearance of the lower portion of the left thorax attracted our attention and suggested strongly the probability of a hernia of the diaphragm. A study of the stereoscopic plates supported this suspicion, and it was deemed important to establish the position of the stomach, for it seemed likely that the dark shadow below the abnormally placed curved line was due to gas in the stomach. With this end in view an emulsion of subcarbonate of bismuth and acacia was given the patient by mouth and a second set of plates made immediately, the patient being in the vertical position (Plate XIII). (Giffin.)

PLATE XIII



Radiographed immediately after the ingestion of bismuth subcarbonate emulsion. Compare with Plate XII.

Note the line of demarcation between the white dense area and the dark area lying above it. The white shadow represents bismuth in the stomach, the upper border of the shadow representing the surface level of the emulsion. Above the bismuth is seen the gas-bubble of the stomach. This is limited above by the wall of the stomach, which we recognize as the curved line described in Plate XII. Note the faint outline of the bismuth lining the esophagus. This can be traced upward to the limit of the plate. Plates XII and XIII, together with the history, confirmed the diagnosis of hernia of the diaphragm.

Important diagnostic points demonstrated by the radiographs, Plates XII and XIII: (1) the presence of a dark circumscribed shadow situated above the indistinct outline of the diaphragm on the left side and bounded above by a curved dense band which does not maintain the dome shape typical of the normal diaphragm line, and limited mesially by the left border of the heart, which is displaced strongly to the right; (2) the presence of the shadows of the lower branches of the left hilus showing through rarefied area; (3) the location of the stomach and its great bubble by bismuth ingestion method. (Giffin.)

or duodenal ulcer. The history of the case, leukocytosis, with relative increase in the polynuclears, and the demonstration of a high diaphragm line lying above the lesion, are the chief points to be relied upon for diagnosis.

Prognosis.—The prognosis is exceedingly grave. Death may result early in infancy as described in the symptomatology of the congenital form, or delayed until adult years. Traumatic cases may die of shock from the causal injury or a fatal issue may not result until years afterward from strangulation of the herniated viscera. Perforation may be the terminal event. The only hope lies in the early diagnosis of the condition before the appearance of urgent strangulation symptoms and in closing the cleft surgically. This has of late been done with brilliant results. When strangulation symptoms appear it is improbable that surgery can prove of much avail, although an emergency operation would naturally be indicated.

Treatment.—The treatment of diaphragmatic hernia is essentially surgical. An attempt should be made to replace the herniated viscera and to close the rent. If the orifice be small, the edges may be sewed together; if larger, the stomach may be sutured to the edges of the aperture so as to close the opening. Serious radical defects, such as congenital absence of half the diaphragm, are of course difficult or impossible of repair. Two exploratory routes have been recommended, a thoracic and an abdominal. Those who favor the thoracic incision claim that the diaphragmatic rent is more accessible for suturing, and that by the entrance of air into the pleural cavity the negative or suction power of the thorax is abolished, so that replacement of the viscera through the cleft is easily accomplished.

The advocates of the abdominal incision are of the opinion that the aspiration or suction power of the thorax is rarely sufficient to prevent reduction by a reasonable amount of traction, and that strangulations are better seen and cared for by abdominal incisions than by the thoracic route. If at any time in the operation it seems advisable, a thoracic incision may be made and the surgical work done by a combination method.

Rare Forms of Diaphragmatic Hernia.—A partial form of diaphragmatic hernia occurs when a pouch-like projection of the stomach wall enters the diaphragmatic cleft. Only two instances of this partial or so-called "Richter's hernia" have been recorded, and in neither case is there any clinical history. One recorded by Andrew is quoted by Knaggs.¹

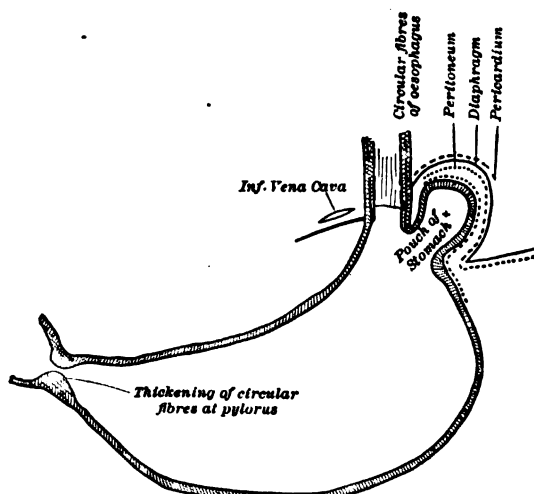
"A dome-shaped pouch of the diaphragm existed immediately to the

¹ Lancet, August 6, 1904.

left of the esophageal opening and into it a portion of the cardiac end of the stomach measuring vertically about one inch was drawn. The hernia could easily be pulled down and the part of the stomach wall that lay against the neck of the pouch was thickened."

In the other instance a pouch formed from the greater curvature had passed through an aperture in the diaphragm and had become strangulated.

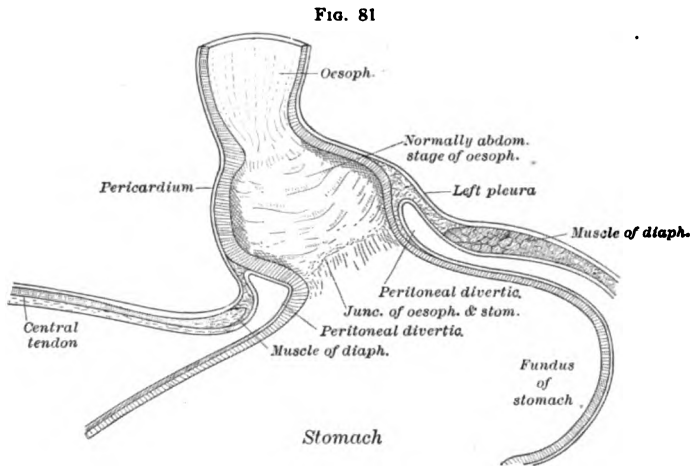
FIG. 80



Stomach showing diverticulum of cardiac end in the pouch of the diaphragm. (From the *Lancet*, March 21, 1903.)

Diaphragmatic Hernia with Gastropnoxis.—Examples have occurred of extreme degrees of visceroptosis in which, owing to the descent of the diaphragm by the giving away of its support, the diaphragm at the seat of the esophageal opening sags downward to a greater extent than can be overcome by a downward traction of the esophagus, so that as the diaphragm descends the esophagus pulls up the cardiac orifice of the stomach to such an extent that this portion of the organ lies within the thorax and above the level of the esophageal aperture. Whether or not such a condition would give rise to clinical symptoms is a matter of conjecture. The writer has encountered one instance of esophageal diverticulum with the characteristic symptoms of cardio-spasm, in which obstruction to the passage of food from the esophagus to the stomach was apparently caused by a redundancy of mucous membrane at the mouth of the sac, causing a valve-like closure of the aperture. Permanent cure was effected by the Mikulicz's operation done by Dr. Erdman, consisting in the opening of the stomach and

stretching the esophageal orifice of the diaphragm by forcible dilatation from below. It is not improbable that this case is an example of this form of hernia.



An example of diaphragmatic hernia of the stomach in a case of ptosis. (From the Proceedings of the Anatomical Society of Great Britain and Ireland, and published in the *Lancet*, March 7, 1903.)

EVENTRATION OF THE DIAPHRAGM

The essential lesion in eventration is the thinning and consequently weakening of the diaphragm, almost invariably on the left side only, so that yielding to intra-abdominal pressure it bulges upward to form a sac into which one or more of the abdominal viscera, particularly the stomach, may enter. The condition is quite distinct from diaphragmatic hernia, as there is no solution of continuity in the diaphragm, and the dislocated viscera are covered by a true sac consisting of the thin and weakened dome of the diaphragm covered on each side by parietal peritoneum. The process in the majority of the recorded instances is evidently congenital; the muscular fibers are scattered and in places absent, so that the diaphragm is thinned and often translucent. Lack of development of the left lung has complicated a number of the recorded cases, and this hypoplasia of the lung points toward the congenital origin of the lesion.

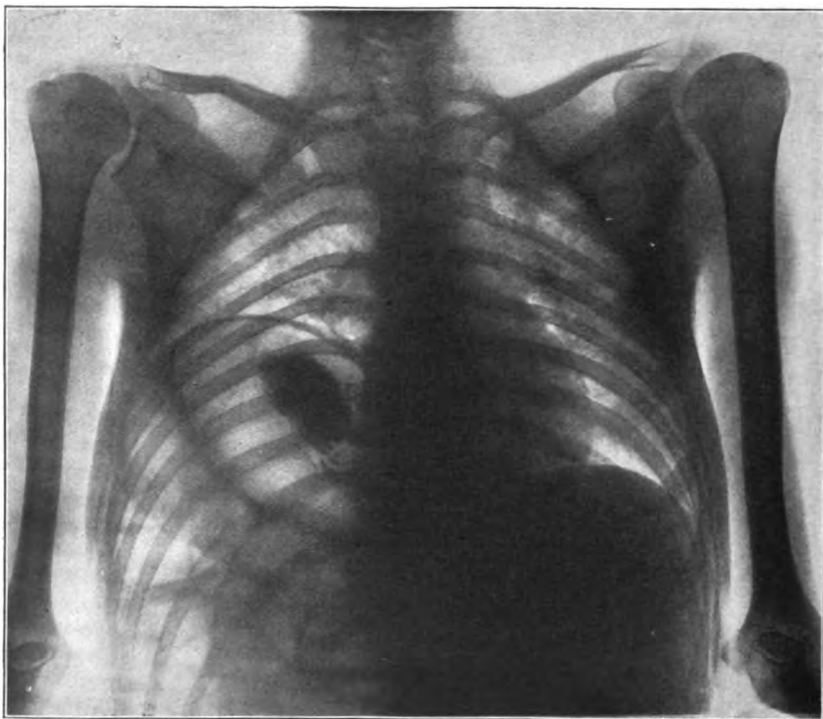
In Sailer's case¹ the left lung was half its normal size. Doering² reports a case in which the left lung was small, but consisted of three

¹ Amer. Jour. Med. Sci., 1905, cxxix, 689.

² Deutsch. Archiv klin. Med., 1902, lxxii, 407.

lobes. Lawrence¹ describes an autopsy where the diaphragm on the left side reached the second rib, forming a sac which contained a greatly dilated stomach. The left lung was reduced to the size of a fist.

FIG. 82



This skiagram was taken with the patient's back to the plate. It is, therefore, as if the patient were standing with his back toward the spectator. On the right side is seen the heart, and below that the convex upper surface of the liver, indicating the position of the right half of the diaphragm. On the left side there is, next to the spinal column, a narrow band of shadow indicating the left border of the heart. Then arising from the vertebral end of the seventh rib, arching to the left and upward as far as the fifth rib and axillary line, there is a convex shadow indicating the position of the left half of the diaphragm. The large dark mass just below this was found at the autopsy to be a huge coagulum of milk lying in the stomach. Below this the attachment of the diaphragm can be seen, represented by a broader line convex on the upper surface, and below this and to the left of the vertebral column a lighter shadow, indicating the position of the spleen. (Sailer and Rhein, radiograph by G. E. Pfahler.)

Eventration may be acquired during the adult life by atrophy or degeneration of the muscular fibers of the diaphragm or by paralysis of the phrenic nerve. Widenman² reports a case of eventration in which the diagnosis was satisfactorily made by the *x*-rays. The left side of

¹ *Lancet*, 1852, ii, 327.

² *Berlin. klin. Woch.*, 1901, ii, 279.

the diaphragm was very high and the stomach lay below the diaphragmatic shadow. The patient died a year afterward of cancer of the tongue (reported by Glaser), and at the autopsy there was found a fatty degeneration of the muscular fibers of the diaphragm caused by a pseudohypertrophic lipomatosis. Stockton¹ reported a case of paralysis of the phrenic nerve in which there was a high position of the diaphragm and tympany in the lower portion of the left thorax. The patient made a rapid recovery, indicating that the lesion was transient and not congenital. The disease has been found in the fetus and the newborn infant. In the remaining cases the ages range from nineteen to seventy-five.

Symptoms.—No characteristic symptoms were present in any of the recorded cases.

Physical Signs.—The physical signs closely resemble those of diaphragmatic hernia, so that a differentiation is often made with extreme difficulty. Tympany is present in the lower portion of the right chest, with feeble or absent breath sounds. Over this portion bubbling, gurgling, and splashing sounds are heard, especially after the patient has swallowed water. The heart is almost regularly displaced to the right. Retraction of the epigastrium and rigidity of the upper abdominal wall do not occur. The diagnosis can only be made with certainty by the *x*-ray. Radiographic plates show a high unbroken diaphragmatic line overlying the boundary of a distended stomach. (See illustration from Sailer.) The differences between the radiographs of eventration and diaphragmatic hernia are given in full under Diaphragmatic Hernia.

Treatment.—There is no treatment, medical or surgical, for the disease. It is this that makes it important for us to differentiate eventration from diaphragmatic hernia.

VOLVULUS

Volvulus of the stomach consists in the abnormal rotation of that organ on one or more of its axes, so that one or both of its orifices become occluded. We recognize a partial and a complete form. In the partial volvulus, one orifice alone is occluded, more frequently the pylorus. It is probable that in many cases the partial volvulus precedes the complete form, the pyloric portion being first occluded, while the cardiac orifice remains patent. This explains the occurrence of vomiting in the earlier stages and also the possibility of passing a tube into the

¹ Buffalo Medical Journal, 1898, xcix, 97.

stomach and successfully performing lavage. When by reason of an increased rotation of the stomach, the cardiac orifice becomes also twisted to the point of occlusion, the volvulus then becomes complete, so that neither vomiting nor the passage of a tube into the stomach becomes possible. The rotation usually occurs on the long axis of the stomach, which passes from the cardia to the pylorus.

Mechanism of Volvulus.—The rotation around the long axis is usually from below, forward, and upward, being aptly compared to the motion of the spokes of a receding wheel. This is known as the anterior volvulus, and it is far more common than the posterior form, in which rotation occurs from below, backward, and upward, following the line of motion of the spokes of an advancing wheel. The cause of the increased frequency of the anterior form is, that it is an accentuation of the normal position of the stomach when it is distended. Inflation of the normal stomach frequently causes the organ to rotate on its cardiopyloric axis, so that the lower curvature passes forward and upward, while the lesser curvature passes downward and backward.

Simmons¹ examined post mortem 50 bodies of infants in which the stomach had been distended, and found that in 40 the lower curvature was directed forward, and the posterior wall was directed downward, so that it became the most dependent portion of the organ. He found, moreover, that the greater the amount of distention in the transverse colon, the more pronounced was the rotation of the stomach.

It is believed by many that the anterior form of volvulus is favored by the natural lines of peristalsis, while the posterior form occurs only in cases in which a reversed peristalsis has occurred. The term *volvulus peristalticus* has therefore been applied by some authors to the anterior form (*i. e.*, the “retreating wheel type”), while the term *volvulus anti-peristalticus* has been applied by others, as Delangre and Neumann, to the posterior or “advancing wheel type.” These terms, however, are but rarely employed.

If the gastrocolic omentum is of normal length and density, the transverse colon is usually dragged up by the volvulus so as to lie above the stomach, being frequently compressed between the stomach and the liver. As the stomach lies in this form of volvulus below the colon, the term *infracolic volvulus* is frequently employed by the German writers. If, however, the gastrocolic omentum is unusually long and relaxed, or if the volvulus is not of an extreme degree, the position of the colon is unaltered and it occupies its normal position beneath the stomach. As the stomach lies thus above the colon, the term *supra-*

¹ Ueber Form und Lage des Magens unter normalen und abnormalen Bedingungen mit zahlreichen photographischen Aufnahmen an Leichen, Jena (G. Fischer), 1907.

colic volvulus is used to designate these cases. The infracolic form is more common in the cases of volvulus in which rotation occurs about the long axis of the stomach, while the supracolic form is more frequent in those cases in which the rotation occurs about anteroposterior, or a vertical axis.

Direction of Rotation.—Anterior volvulus about the long axis of the stomach is the ordinary form of volvulus associated with diaphragmatic hernia, and with non-malignant tumors of the stomach.

Rotation of the stomach about its *vertical* axis is much less frequent. The usual line of rotation is “contra clock-wise,” when viewed from the head of the patient, the pyloric portion of revolving forward and from right to left. This form is almost exclusively a complication of hour-glass contraction of the stomach. (Cases of Langerhans, Mazzotti, Saake.)

Rotation about the vertical axis, in a “clock-wise” direction in which the pyloric portion moves backward and from right to left has occurred only in one known case (Berti¹). It may be said, however, that the descriptions of many of the cases of volvulus are either so involved, or so inadequate, that it is frequently impossible to determine with any certainty the exact lines of rotation, especially as in some instances rotation on several axes have occurred in the same case.

Rotation about an *anteroposterior* axis is the rarest form, and but one case (Streit²) is recorded. In this instance the stomach twisted itself on its anteroposterior axis, “contra clock-wise,” the pyloric portion revolving downward and from right to left.

The Degree of Rotation Varies.—It is very probable that slight degrees of volvulus occur, causing temporary pain distention and vomiting, and that the emptying of the stomach by the emesis is followed by a spontaneous reduction to the normal position. In these cases the volvulus can be partial, involving the pyloric orifice, while the cardiac orifice remains patent. While in many of these cases spontaneous reduction occurs, others undergo a greater degree of torsion and produce the complete form from which spontaneous recovery is practically impossible.

In the majority of developed cases the volvulus is one of 180 degrees, so that the stomach lies completely upside down.

In rarer cases more extreme degrees have occurred. Pendl³ reports a case in which rotation of 270 degrees occurred on the longitudinal axis, while in Berti's case a double twist occurred.

¹ *Gaz. med. ital. prov. Venete, Padova*, 1866, Bd. ix, pp. 139 to 141.

² *Amer. Jour. Med. Sci.*, 1906, cxxxi, 967.

³ *Wien. klin. Woch.*, 1904, No. 17, p. 476.

Berti's¹ case is as follows: Female, aged sixty years, began two hours after a hearty dinner with severe pains in the stomach and vomiting, followed by abdominal distention. The case was seen twenty-two hours afterward in profound collapse.

Autopsy.—The distended stomach occupied nearly the entire abdominal cavity, and had undergone a double turn about its longitudinal axis, occluding both orifices, and by the extreme torsion bringing the pylorus and cardia into juxtaposition. Two complete turns from right to left were required to reduce the volvulus.

The transverse colon was compressed between the stomach and liver, while the spleen and pancreas were displaced together into the pelvis. The spleen had undergone two revolutions on its axis from left to right.

Effect of Volvulus.—The effect of volvulus upon the stomach is practically that of strangulation. The stomach becomes congested, its walls thickened and edematous. In the majority of instances the whole organ is covered by the omentum, which has become fixed by recent adhesions. There is great distention, the stomach often apparently occupying the entire abdominal cavity. This is due to the pouring out of fluid into the cavity of the stomach, usually to the amount of several liters. In Wiesinger's case 4 liters were evacuated by aspiration before the organ could be replaced, while amounts of 3 liters have been frequently encountered. The fluid is usually dark brown in color, from the presence of altered blood; more rarely greenish in tint.²

In Pendl's case, lactic acid was present, with an absence of hydrochloric acid.

In Cordier's³ patient the stomach was filled with gas and offensive material, fecal in character. The matters vomited during life had been distinctly feculent. As in this case total and complete volvulus had occurred, complicating a diaphragmatic hernia; no logical reason for the fecal nature of the fluid can be given.

The mucous membrane of the stomach is congested and shows multiple punctate hemorrhages; in some instances the mucous membrane is of a dark brown color, as if it had been painted by an escharotic.

Erosion of larger or smaller areas of mucous membrane may occur, or even areas of gangrene. Death usually results, however, before gangrene becomes marked.

Perforation of the stomach may occur either from traumatism at the time of the volvulus or later from the perforation of softened necrotic

¹ *Gaz. med. ital. prov. Venete*, Padova, 1866, Bd. ix, pp. 139 to 141.

² *Nord Med. Ark*, Stockholm, 1897, N. F., Band viii, No. 19.

³ *Annals of Surgery*, September, 1897, p. 353.

areas. As an example of perforation from rupture may be cited the case of Collischon,¹ who died with symptoms of a high-seated obstruction. Autopsy showed the liver displaced downward, lying in the right lower section of the abdomen. The lesser curvature of the stomach was adherent in its middle to the under surface of the left lobe of the liver, and had been dragged downward and forward, producing a volvulus of the long axis, with a tear of the stomach wall at the site of the adhesion.

Peritonitis may occur without perforation, and may either be localized or general. In volvulus complicating diaphragmatic hernia, pleurisy and septic pneumonia may result.

Torsion and injury to other abdominal viscera may occur at the time of volvulus. A case is reported by Wilke.²

There may be tears in the lesser omentum (Berg), rupture of the splenophrenic ligament,³ or even complete separation of the entire greater omentum.

The spleen may be ruptured either by traction on or torsion of the gastrosplenic ligament,⁴ or it may be twisted, as in Berti's⁵ case, upon its axis.

Etiology.—It is convenient in the description of the etiology of volvulus to divide the cases into five clinical groups, and to describe each type separately.

I. Volvulus and Diaphragmatic Hernia.—Diaphragmatic hernia has been elsewhere described, so that in this connection only the mode of origin of the complicating volvulus will be given.

Volvulus complicating diaphragmatic hernia may occur in one of two ways, causing either a partial or a complete occlusion.

In the partial form the middle or the pyloric region of the stomach passes into the thoracic cavity through the hernial cleft, leaving the fundus and cardiac portion on the abdominal side of the diaphragm. As the hernial orifice is usually at or near the esophageal opening the entrant portions have to rotate upward and to the left, so that the pylorus comes to lie near the cardiac orifice, producing thus a rotation about the anteroposterior axis. The pylorus becomes occluded, but the cardiac orifice is patent.

In many of these cases the herniated portion undergoes no change and can be easily withdrawn, while the cardiac portion within the abdominal cavity suffers from the effects of strangulation.

¹ Inaug. Dissert., Kiel, 1888.

² Münch. med. Woch., 1907, liv, No. 20, p. 969.

³ Dini, quoted by Payer, Mitteil. a. d. Grenz. d. Med. u. Chir., Band xx, Heft 4.

⁴ Borchardt, Arch. f. klin. Chir., Band lxxiv, Heft 2.

⁵ Loc. cit.

In the second variety the volvulus is complete, both orifices being occluded. Through the diaphragmatic opening, more or less of the stomach passes with the greater curvature uppermost; the larger the opening the greater the entrant portion and the more pronounced the degree of volvulus. In many cases the whole stomach has been found in the hernial sac, rotated on its long axis. In these cases it is generally supposed that the greater omentum is the first to enter, dragging after it the greater curvature and then the rest of the stomach.

Such an instance is given by McClosky.¹ A woman, aged twenty-two years, entered the hospital complaining of vomiting, pain in the left chest and back, passed into collapse, and died. Duration of illness two days. Autopsy showed a diaphragmatic hernia of the left side, consisting of the entire stomach, first part of the duodenum, omentum, spleen, and part of the small intestine. The greater curvature was uppermost. The stomach was dilated and contained dark red bloody fluid; its mucous membrane was congested and eroded in places, but was not gangrenous. The heart was displaced to the right. The left lung considerably pushed upward against the clavicle, occupied only the supra- and infraclavicular regions.

II. Volvulus and Tumors.—Tumors of the stomach may cause volvulus by reason of their weight, provided that they are situated near the lesser curvature and are not adherent to neighboring parts. Sarcoma and carcinoma are not commonly the cause for such displacement, owing to their tendency to form adhesions before they arrive at the requisite size and weight; whereas, the benign tumors fibroma and fibromyoma often grow to a weight sufficient to cause tilting before the fixation of the stomach by adhesion occurs.

Kaufmann² describes a case in which a fibromyoma weighing 2325 grams, attached to the lesser curvature, had caused a volvulus with a descent of the stomach so that it was mistaken for a tumor of the ovary. In this case death resulted from strangulation symptoms, although in other cases the axial torsion has been so gradual and incomplete that characteristic clinical symptoms have been conspicuously absent.

In a patient of v. Hacker³ a fibromyoma the size of a man's head, and attached to the lesser curvature, had produced a partial incomplete volvulus, while in Erlach's⁴ case a myoma weighing five and a half kilos was removed by operation, relieving a similar torsion of the stomach. In neither of these cases were gastric symptoms marked.

¹ *Lancet*, May 4, 1895.

² *Lehrbuch der speziellen Pathologischen Anatomie*, 1907, p. 421.

³ *Wien. klin. Woch.*, 1900, No. 6, p. 146.

⁴ *Ibid.*, 1895, No. 15, p. 272.

III. Volvulus and Hour-glass Stomach.—Volvulus complicating hour-glass contraction of the stomach has occurred in a small number of cases. The torsion is regularly partial and involves the portion of the stomach between the pylorus and the constriction. Its occurrence is favored by the presence of single bands of adhesions at the point either of the constriction or of the pylorus so as to form a fixed point.

An example of this form is given by Langerhans.¹ A woman, aged forty-seven years, who for years had suffered from serious stomach trouble, was suddenly seized by vomiting and severe abdominal pain. Upon her entrance to the hospital, with the diagnosis of benign pyloric stenosis, the stomach was repeatedly washed, each time showing the presence of blood in the stomach contents. Extreme thirst, anuria, and finally tetany preceded the fatal issue.

Autopsy revealed a thick band-like adhesion proceeding from the lesser curvature, passing to the anterior abdominal wall in the left parasternal line, causing a constriction of the stomach just sufficient to admit the passage of one finger.

The volvulus being partial and the cardiac orifice patent, the swallowing of food, vomiting, and the performance of lavage were all possible.

IV. Volvulus and Trauma.—Volvulus may be caused by the traction on the stomach by sudden or traumatic displacement downward of other abdominal organs, especially the liver and spleen.

Collishon's case has been previously described in which the liver was suddenly displaced downward, dragging after it the stomach and causing a rotation on the long axis by reason of adhesions between the under surface of the left lobe and the middle of the lesser curvature, and causing a tearing of the gastric wall at the site of the adhesion.

Dini² reports a case of a peasant woman, aged forty-two years, who for a long time had suffered from pain in the stomach, which was interrupted by long intervals of freedom. One day, after eating, she worked in a bent-over position and experienced a sudden pain in the stomach, more severe than she ever had before, and began to vomit. She entered the hospital in collapse and died on the fourth day of her illness.

Autopsy.—An enlarged spleen lay behind the symphysis; the spleno-phrenic ligament was torn across. By traction on the gastrosplenic ligament the cardiac portion of the stomach was drawn downward, resulting in an anterior volvulus.

V. Idiopathic Cases.—There is a group in which no one of the above-mentioned causes can be assigned, and to which the unsatisfactory

¹ Virchow's Arch., 1888, cxi, 387.

² Quoted by Payer, Mitteil. a. d. Grenzgeb. d. Med. u. Chir., Band xx, Heft 4.

name of idiopathic volvulus is given. In the majority of these cases the symptoms appear after a full meal, showing that distention of the stomach has something to do with the reactions.

In other cases there is a history of some distinct traumatism, either a fall or a blow on the stomach.

Payer's case¹ is thus reported. A male, aged fifty-nine years, fell from a ladder an hour and a half after dinner, landing on his feet with a very considerable jar, and immediately felt a terrible pressure in the middle of his stomach and over his heart. He passed into collapse and was admitted into the hospital an hour after the accident. Operation seven hours after his injury, showing the stomach wrapped about by the omentum, and a rotation of 180 degrees, with the colon lying between the stomach and the liver. Borchardt's case² is a man, aged forty-four years, who was hit in the stomach by a great iron key and was operated on two days later, but died of hemorrhage from the stomach incision.

The autopsy showed an aneurysm of the aorta and a volvulus of the stomach of 180 degrees on its horizontal axis, with occlusion of both orifices, and the colon lying underneath the stomach.

In other cases the symptoms come on without previous traumatism and the cause for the volvulus is obscure.

The majority of writers who have described these cases, consider that the predisposing cause for volvulus is a gastrop-tosis, with the attendant laxity of the supporting ligaments of the stomach, and that the exciting cause might be any sudden increase of abdominal pressure, such as straining, lifting, or coughing, and that, furthermore, the upward lift of the stomach is favored by distention of the colon. Gastrop-tosis is so frequent, however, and volvulus is so rare that it seems difficult to believe that there is a causal relation between the two.

Symptoms.—In many instances amounting to about one-half of the total number of the cases reported the symptoms of hour-glass stomach, diaphragmatic hernia, or of gastric adhesions precede those of the actual volvulus.

On other cases the symptoms begin abruptly and are fairly characteristic. Pain is a constant symptom and is not only continuous, but is of the greatest intensity and quite unlike any pain that the patient has ever before experienced. It is located usually in the epigastrium and left lower thorax, although in cases of volvulus associated with descent of the stomach, the pain may be located lower down in the abdomen. There is a peculiar form of pain often observed, especially

¹ Mitteil. a. d. Grenzgeb. d. Med. u. Chir., Band xx, Heft 4, S. 708.

² Arch. f. klin. Chir., Band lxxiv, Heft 2.

in volvulus, associated with diaphragmatic hernia, which was described by Faure and by him designated the "*douleur thoracique*." This consists in a pressure feeling over the heart as if the thoracic box was squeezed together with such force that it was about to break, and is in fact a pressure symptom induced by a distended and high-lying stomach. Faure considered that local peritonitis in this neighborhood of the stomach had a great deal to do with this form of thoracic pain.

Vomiting usually marks the onset in the stage of partial volvulus. As long as the cardia remains patent vomiting is possible, food being usually rejected soon after its ingestion. During this stage the stomach-tube can be readily passed its entire length and the stomach can be successfully washed.

There are undoubtedly cases characterized by pain and vomiting and slight shock which are due to partial incomplete volvulus and which subside either spontaneously or after the emptying of the stomach by vomiting or lavage. It is often impossible to differentiate this condition from the temporary enlargement of the stomach in a diaphragmatic hernia with spontaneous reduction.

In the great majority of instances, however, reduction does not take place, but the volvulus becomes complete, with occlusion now of both orifices. When it occurs, vomiting and the passage of a tube into the stomach become impossible. Retching with severe effort, but without result becomes more and more frequent, thirst becomes more and more agonizing, and any liquids taken are immediately rejected without any admixture of gastric contents.

When this clinical picture is present, with the characteristic physical signs and the impossibility of passing a stomach-tube through the cardiac orifice, the diagnosis is one of no great difficulty. These symptoms may last from a few hours to as many as fourteen days before passing into collapse.

Payer's case of a man who after a hearty dinner fell from a ladder, landing with a shock on his feet, and who immediately suffered from pain and vomiting, was admitted to the hospital one hour later. Operation was unsuccessfully performed seven hours after the injury.

Collishon's case previously alluded to did not enter the hospital until the fourteenth day.

Collapse is usually profound and is usually attended by some instigation of the pain and efforts of vomiting.

Death results from collapse, usually between the second to fourth day of the disease; more rarely between the seventh and fifteenth day. The fatal issue may be hastened by perforative peritonitis, septic peritonitis without actual perforation, intestinal hemorrhage or from other lesions of severe traumatism.

Diagnosis.—Physical Examination.—The most constant physical sign of volvulus is a spreading tympanitic distention of the area occupied by the stomach. In the majority of instances this distention appears in the left hypochondrium and slowly extends, so as to occupy more or less of the entire abdomen, the remaining portions of which are ordinarily soft and insensitive. It is usually possible to make out by palpation the balloon-like form of the distended viscus.

If the stomach be displaced downward the tympanic tumor may lie in the lower abdominal region, as in Newmann's case, where the stomach was palpable below the navel, and in Hermes' case, where it was found in the middle of the abdomen. If the volvulus complicates a diaphragmatic hernia the physical signs are those of the latter condition and will be described under that heading.

Localized rigidity of the abdominal muscles may appear early in the disease, and is apt to spread as the disease progresses. Should peritonitis or perforation occur, the rigidity becomes more intense and generalized.

The heart is usually displaced in the form of volvulus associated with diaphragmatic hernia and dextrocardia is the rule. If the volvulus, however, be below the diaphragm the heart is more apt to be displaced to the left, although dextrocardia in this form is not infrequently observed.

Very little difficulty should be observed in the diagnosis of volvulus, if the symptoms are well pronounced. The most common errors in diagnosis have been in mistaking volvulus for ruptured ulcer of the stomach, for intestinal obstruction or for pneumothorax.

In perforation of gastric ulcer, we have the sudden onset of pain and occurrence of shock, as in volvulus, but the abdomen is retracted and board-like, whereas in volvulus we have the physical signs of a localized spreading of tympany, with but slightly localized rigidity. But little harm is done, however, by mistaking these conditions, as in each the treatment is surgical.

The differential diagnosis between volvulus associated with diaphragmatic hernia and pneumothorax will be considered under the heading of Diaphragmatic Hernia.

Volvulus is to be differentiated from a high-lying intestinal obstruction by the absence of intestinal vomiting, and finally by the absence of vomiting altogether in the later stages, and also by the inability to pass a stomach-tube through the cardiac orifice. The physical signs of the spreading area of tympany with resistance in the left hypochondrium are not observed in the intestinal obstruction cases.

Hemorrhage into the pancreas should be thought of in all cases of sudden and severe abdominal pain and shock if the abdomen is sunken

and rigid, quite different from the localized and spreading tympany of volvulus.

Acute dilatation of the stomach or "arteriomesenteric ileus" gives rise to physical signs more or less identical with those of volvulus, but the onset is not quite as acute, and the stomach-tube may be passed easily into the stomach, although it must be remembered that with lavage in cases of acute dilatation the return flow may be difficult on account of the inability of the stomach to contract sufficiently to allow its contents to be expelled.

Prognosis.—Partial incomplete volvulus is not incompatible with spontaneous reduction and cure. These cases are, however, rare compared with those in which the volvulus is complete. When volvulus once becomes well-developed, recovery without surgical intervention is well-nigh impossible.

Treatment.—As soon as the diagnosis is made, an attempt should be made to pass a stomach-tube and to wash out the stomach, hoping that by the withdrawal of the stomach contents, both fluid and gaseous, the twisted organ may be able to revert to its normal position. Failing to accomplish this result no time should be wasted, and immediate operation should be performed. Payer's case was operated on seven hours after the injury and unsuccessfully. A number of patients have been operated on two or three days after the onset of the symptoms, with excellent results, but it must be remembered that the longer the case goes without operative intervention the greater the tendency toward fatal issue.

CHAPTER XVI

GASTROPTOSIS

THE term "gastroptosis" signifies literally a descent of the stomach, so that it assumes a lower level than normal in the abdominal cavity. Enteroptosis and splanchnoptosis are the terms used indicating a downward displacement of the abdominal organs generally, the stomach as well as the other viscera. These terms, however, are usually exchangeable, as gastroptosis rarely occurs alone, but is usually associated with general visceral descent—while in the visceral ptoses in general the stomach assumes its share in the process.

The terms "gastroptosis" and "splanchnoptosis" though not as etymologically correct as "gastroptosis" and "splanchnoptosis" are nevertheless sanctioned by long usage, and are therefore used by the writer.

Frequency and Occurrence.—The disease is exceedingly common. The writer finds that in private practice one patient in every six who applies for relief from gastro-intestinal symptoms, presents evidence of this complaint, although not always to its full degree of development. German writers state that it is more common in hospital than in private practice. Elsner writes from Berlin that the disease is far more frequently seen in the working classes than in those well-to-do, and attributes this frequency to insufficient nourishment and hard physical work. The writer's experience in New York is quite the reverse of this. Gastroptosis is of every-day occurrence in private practice, but is far less commonly seen in hospitals, so much so that in his clinics it has often been difficult to find a case for demonstration.

The symptoms may appear at any age after puberty, but usually become noticeable during the early portion of the storm and stress period of life. The manifestations of the disorder may then be continued throughout life and into the period of old age. Their first appearance is rarely deferred until after the fiftieth year. Women are more frequent sufferers than men in the proportion of 7 to 1. This predisposition to ptosis in women is often ascribed to the natural lack of muscular development of the abdominal wall, too frequent child-bearing or tight-lacing. The explanation that seems most plausible to the writer is that the more sensitive nervous organism of women and their greater predisposition to congenital and acquired

neurasthenic conditions render them more liable to the complaint. The enteroptotic habitus is said to occur in about 25 per cent. of women, a far greater proportion than that observed in men.

Etiology.—Two distinct causes for visceral ptosis are described—a congenital form, due to inherent physical weakness, and an acquired form, due to relaxation of the muscular wall of the abdomen, or to the mechanical displacement of the stomach downward by tight-lacing.

Acquired Form.—Glenard, to whom we owe our first observations of the disease, considered that the starting-point of the condition was the weakening of the hepaticocolic ligament, allowing a falling of the hepatic flexure of the colon, followed by a relaxation of the other ligaments and mesenteries and a dropping downward of the viscera dependent upon them for support. He attributed this relaxation to a constitutional defect peculiar to certain individuals by which the strength of the supporting power of the ligaments and mesenteric tissues are insufficient to support their normal weight.

Landau considered that the primary cause lay in an acquired weakness of the abdominal wall, giving as an example the ptosis commonly observed after childbirth. To the visceral displacement resulting from extreme relaxation of the abdominal muscles the name of "Landau's Enteroptosis" has been applied.

Meinert considered gastroptosis to be a frequent accompaniment of chlorosis in young girls, but Meinert's deductions are probably inaccurate, as he diagnosticated the condition whenever he located the lesser curvature of the stomach below the umbilicus by forcible distention of the organ. The lower border of any atonic stomach that is artificially overdistended may lie too low in the abdominal cavity, and it is most probable that many of Meinert's cases were those of atony rather than of gastroptosis.

Keith who bases his theories upon anatomical rather than upon clinical studies, believes that enteroptosis is the result of vitiated methods of respiration. According to this writer, the organs within the thoracic and abdominal cavities are poised between the muscles of inspiration and expiration and swing with each respiratory tide. In the majority of people this respiratory ebb and flow are so finely adjusted that the changes in the location of the viscera do not occur. In other cases, the balance between inspiration and expiration may be upset and the condition of enteroptosis produced. Keith's conclusions are thus epitomized by Brown:

"1. The contraction of the diaphragm is a factor which produces a displacement of the viscera in splanchnoptosis or Glenard's disease, and further, that of the various parts of the muscles the crura are the most important agents in producing this result.

"2. Before this displacement can be produced, either what he terms the thoracic supports of the diaphragm must have yielded or the antagonistic abdominal muscles must have been hampered and weakened in their action, as in the example of tight corsets.

"3. The bands which fix the viscera to the wall of the abdomen are of quite subsidiary importance. Displacement of the liver and stomach arises chiefly from two causes: relaxation or paresis of the abdominal muscles which maintain the visceral shelves, or more frequently constriction of the body cavity by clothing or disease, so that the normal respiratory swing forward cannot take place."

Keith's paper is so full of interesting details that even this well-worded epitome fails to give a clear idea of the many important observations made by him in his anatomical studies of this disease, and, therefore, the reader is referred to the original article in the London *Lancet*, March 7 and 14, 1903.

In recent times Rosengart by a study of fetal development is led to believe that gastropptosis is due to a lack of postnatal development or to a reversion toward the embryonic type. In the fetus the position of the stomach is vertical, and the liver rests upon the superior surface of the kidney. After birth there is ascent of the various viscera due to the rotation of the liver upward, after the beginning of the respiratory act, so that this organ rises to the dome of the diaphragm and relatively undergoes a diminution in size, allowing an upward shift of the neighboring organs. He believes that a downward displacement of the liver or a lack of its ascent either from intrathoracic causes, which press downward upon the diaphragm, or from external pressure over the lower costal arch, or from a weakening of the abdominal muscles, is the chief cause for similar displacements of the neighboring organs and their reversion to the congenital position.

Tight-lacing has always been held responsible for gastropptosis. Rovsing¹ divides gastropptosis into the virginal and the maternal types. The first variety occurs in young girls and is distinctly attributable to their misuse of corsets and laces, while in the maternal type the change in the intra-abdominal pressure due to pregnancy and child-bearing is responsible for the disorder. To this view of Rovsing the writer cannot agree, as it does not seem that tight-lacing has been a factor in inducing the ailment in more than a very few of his patients. Rovsing does not indicate the causes for the ailment in men who have neither worn tight corsets nor borne children. Tight-lacing is if anything more common in those without gastropptosis than in those who are the subjects of this disorder, as the former patients are usually more corpulent

¹ Jour. Amer. Med. Assoc., August 3, 1911.

and therefore more apt to lace themselves tightly. The older form of corset which causes constriction about the waist line as would a tight narrow belt is undoubtedly injurious, hampering diaphragmatic breathing and tending to displace downward the organs in the thoracic abdomen; but the modern straight front corset is free from these objections and is beneficial rather than injurious. In those, however, whose physical conformation is that described by Stiller, with long thorax and low-lying costal arch, clothing that is too tightly belted about the waist may press upon the lower costal arch instead of on a line below the ribs, as in normal people, and may thus displace the entire costal arch so as to diminish the capacity of the thoracic abdomen and cause a downward displacement of the organs normally situated within its confines.

Congenital Form.—The most important contribution to the study of gastroptosis has come from Stiller, who considers that visceral ptoses accompany a peculiar form of physical development which he describes by the term *Habitus Enteroptoticus* or *Habitus Paralyticus*. The thorax is long and narrow, the epigastric angle is abnormally acute and the costal arches run downward and outward in a long more or less vertical sweep that gives a narrow elongated shape to the thoracic abdomen, greatly diminishing its capacity.

Owing to the abnormal development of the thoracic abdomen and to its diminished capacity, the organs contained within its confines have no space to lie transversely, but are forced to assume a more vertical position and to be pushed downward to obtain sufficient room for growth and activity, so that ptosis is obviously bound to occur.

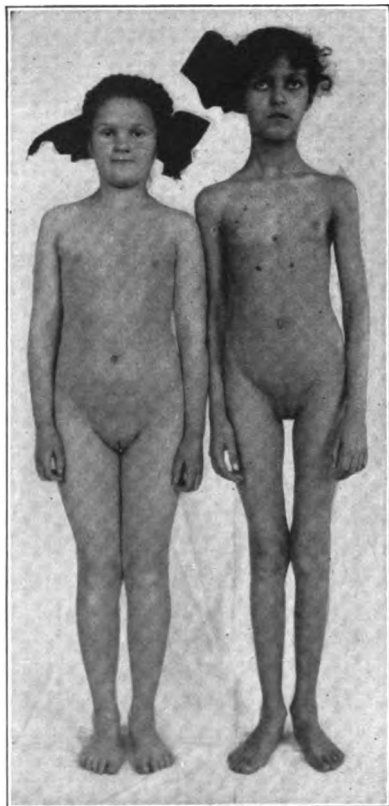
With such a conformation, improperly fitted corsets and tight belt lines constrict the lower arches of the ribs, as they are lower than in other individuals and tend to add to the displacement.

The writer is absolutely in accord with Stiller's views. All mankind may be divided into two classes: those with and those without the enteroptotic habit. Those without the enteroptotic habit, who are possessed of a broad thorax and wide costal angle, are not apt to be neurasthenic. If they suffer from gastro-intestinal distress it is apt to be the result of organic disease. Those, on the other hand, who show the stigmas of the enteroptotic habit, whose abdomen and thorax are long and narrow and whose costal arch is sharp, are constitutionally and temperamentally neurasthenic and are subject to a great variety of functional disorders. When such a patient suffers from gastro-intestinal distress, while there may be an organic cause present, it is fair to assume that a large proportion of the symptoms are neurasthenic or functional in character.

In all such patients as Stiller describes, weakening of the abdominal wall from any cause whatever tends further to increase the natural

tendency to downward displacement and may therefore be regarded as a contributory cause for the fuller development of visceral ptosis. There are, however, a small number of patients in whom the bodily conformation is normal, the thorax is broad, the costal angle wide, and

FIG. 83



(A. B. and E. R.) Two children, aged eleven years. Contrast contour of form, amount of fat, muscular development, bony frame, and size of chest.¹

in whom the downward displacement of the stomach is effected by purely mechanical causes, such as weakening of the abdominal wall, often with or without diastasis of the recti, either the result of frequent pregnancies or due to the removal of large abdominal tumors, or the repeated withdrawal of ascitic fluid. The stomach may be, moreover, displaced by the weight of attached tumors, or dragged by adhesions into a faulty position. In the latter instances, the displacement of the stomach is not accompanied by other visceral ptoses, and does not come within the description of the disease as given in the present article.

Enteroptosis in Children.—Often in children are observed signs of muscular insufficiency and frailness of frame representing the primary characteristics of the enteroptotic habit of the adult. Changes in the shape of the thorax and angle of the ribs are seldom demonstrable until the twelfth year, which is the period marking the transition from the infantile to the adult form of bodily development. Until this age is reached there is very seldom any

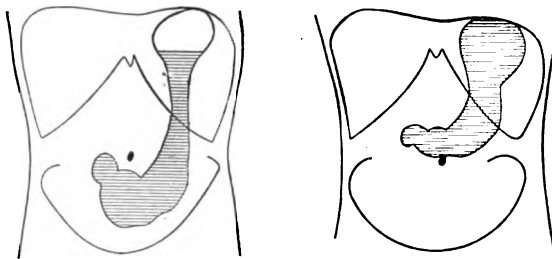
actual descent of the stomach or other viscera. The stomach lies well above the umbilicus until after the twelfth and fourteenth year, although the lower pole may be distinctly hooked.

Position and Shape of the Stomach in Gastroptosis.—From the cardia to the outer extremity of the first inch of duodenum the stomach is

¹ Smith, Jour. Amer. Med. Assoc., 1912, lviii, 391.

surrounded by peritoneum and is freely movable, although the radius of motion of the pylorus is small compared to the body of the stomach. Being swung between these two fixed points as the organ sags it assumes a vertical position terminating in a well-marked upward hook just before the pylorus is reached—at about the line of the incisura angularis,—so that a “looped” or “fish-hook” form is produced. If the pyloric end be fairly well maintained in place, the bend is more pronounced and the ascending arm is more vertical, so that the stomach assumes the “water-trap” or “drain-trap” form. When the duodenal attachments are relaxed, the whole pyloric end sinks until all but about the pyloric region itself lies nearly vertically up and down.

FIG. 84



Ptosis. A, in the vertical position; B, in the horizontal position. (Hertz.)

When food is put into the stomach the lower limit of the organ sags more and more deeply into the abdomen, and the body of the stomach lies more nearly vertical. The tubular form of the body well constricted by normal tonus upon its contents is rarely seen, as the food column is slight in diameter or even entirely absent, so that the outline of the stomach shows a large pear-shaped or bulbous air-bubble at its upper end connected by a collapsed and empty body of the organ, with a lower food chamber which is sagged downward by mechanical weight. In advanced cases of hypotonus there may be a close resemblance to an hour-glass stomach. These changes in outline are clearly evident when the patient stands, but disappear when he lies down. In recumbency the greater curvature may be well above the navel, so that in this position the x-ray may not afford the least proof of any degree of ptosis.

Symptoms.—Gastroptosis often exists without giving rise to any local symptoms, and may run for years an entirely latent course. A displaced stomach will do its work without symptoms of indigestion, unless it becomes atonic, to which condition it is by nature predisposed. Whenever symptoms appear they are regularly due more to the atony

than to the gastroptosis itself. The whole symptomatology and treatment of gastroptosis is that of atony. We describe therefore:

1. Gastroptosis without atony.
2. Gastroptosis with atony.

Gastroptosis without Atony.—In this stage there are few if any gastric symptoms, certainly none that are characteristic or prominent. Neurasthenic symptoms are, however, more or less pronounced, and the patient is apt to be of unstable nervous temperament, easily affected by environment, and without mental or physical endurance. Strains that should not be considered excessive are followed by an abnormal physical and mental reaction.

Nutrition is regularly below par, and it is often a matter of serious concern. These are the patients who are constantly taking tonics, and who are forced to leave home every little while to “rest up” and regain what they have lost by the friction of daily life. The bowels, as a rule, are constipated.

Physical examination almost invariably shows the enteroptotic habitus—the long abdomen and thorax, narrow costal angle, together with evidences of visceral ptoses. The description of the physical signs are given in full detail in a later section.

The early recognition of these cases is of extreme importance, because it must be acknowledged at the start that such a patient is unable to stand as well as others the storm and stress of life, but that they are always more or less delicate and will have to conserve as far as possible their nervous and physical energies all through life. It is, therefore, important to inaugurate a prophylactic treatment that will minimize their liability to succumb by the way.

Gastroptosis with Atony.—Gastroptosis with atony runs a different clinical course in the congenital and in the acquired cases. In the former group the gastro-intestinal symptoms are combined with those of the underlying neurasthenic habit, so as to form a varied clinical picture, while in the acquired type of the disorder the symptoms are those of the gastric ailment itself without the same predominance of nervous phenomena. The following description of the symptoms will apply more definitely to the former group; the difference in the clinical course of the two forms will be alluded to later in the discussion.

If patients with gastroptosis live within their physical and nervous limitations they are not apt to suffer, but if these limitations are transgressed, symptoms of indigestion and of neurasthenia regularly appear. At first the symptoms appear only from time to time, induced by some unwonted physical or mental strain and are but transient, disappearing when the patient regains the vitality that has been lost. Relapses occur, however, so that the symptoms tend to become more or less

continuous, and to persist until radical means are taken to restore nervous and physical tone and to relieve the atonic condition of the stomach wall itself.

In 90 per cent. of the writer's cases the symptoms began after a definite and existing cause. Prolonged nervous strain, sudden mental shocks, grief, depression of spirits, disappointments in love, are the most frequent causes adduced. The history of physical strain is not infrequently elicited; any severe or exhausting illness, such as typhoid fever, or la grippe may be followed by gastric symptoms, or the strain of hard travel, nursing sick relatives, or long business hours may so devitalize an individual with an enteroptotic inheritance as to cause more or less complete invalidism.

In many patients the history of a definite exciting cause may not be obtained, but the clinical history brings out clearly enough that the patient is quite unable to endure the wear and tear of the ordinary demands of life. The history of almost every patient with gastroptosis may be divided into three stages.

1. The stage without atony in which the symptoms are those of subnutrition, neurasthenia, and lack of vitality.

2. A stage of temporary and recurring atony following physical or nervous strain, and accompanied by intermitting symptoms.

3. The stage of permanent atony in which the symptoms are continuous.

An appreciation of these three successive stages of the disease is essential to a rational and intelligent treatment. These stages are well illustrated in the following history, which is quite typical of the course of the ailment:

L. R., a lady, aged twenty-seven years, was a delicate under-nourished child, and though free from serious illness was always taking tonics. She never could lead the life that her companions did without having to rest up for long periods at a time. When fifteen she noticed that from time to time, following physical or nervous fatigue, she suffered from heart-burn, gas in the stomach, and distress after meals. These symptoms disappeared after she rested. When eighteen she entered upon an active social life and did a great deal under high tension. Symptoms now became continuous and severe, she lost weight and became a nervous wreck. The more she dieted the worse she became.

The symptoms of gastroptosis may be divided into four groups:

1. The symptoms of atony.
2. Subnutrition.
3. Neurasthenia.
4. Symptoms due to associated displacements.

1. **Symptoms of Atony.**—The symptoms due to atony in gastroptosis do not differ in the least from those due to atony however induced, except that flatulence is a more constant and prominent symptom and is usually most annoying two or three hours after eating, although it may be most severe during the night or awaking the patient during the early morning hours. Most of the gas is raised, but a considerable quantity remains and may occasion constant distress. The flatulence, as in atony, is influenced by the bulk and weight of the food rather than by its quality, and the patients are very apt to attribute their discomfort to what they eat and reduce their diet to a starvation point without any relief from their distress. Intestinal distention occurs later after eating than does gastric flatulence and is more continuous. Heaviness, a feeling of fulness and weight in the stomach, especially after eating, are prominent symptoms in the majority of instances.

Actual pain is not a common symptom but it occurs more frequently than in the cases of simple atony. The pain may be due to a variety of causes. Painful distention of the stomach may occur from accumulation of gas, and is relieved when gas is raised. It is probable that in many of these instances the cause is to be found either in mesenteric constriction of the duodenum by downward traction of the root of the mesentery and superior mesenteric artery, as the result of prolapse of the intestine, or downward displacement of the small intestine may drag upon the duodenum at certain fixed points, either at the angle of the first and second portion of the duodenum or at the duodenojejunal junction, and produce a certain degree of obstruction.

Robinson,¹ indeed, believes duodenojejunal obstruction is frequently the cause of death in gastroptosis cases over forty years, as he has found 15 or 20 cases of distinct and extensive gastro-intestinal dilatation on the right side of the superior mesenteric bloodvessels. This the writer thinks is an extreme statement not corroborated by clinical observation. These factors are not sufficient in gastroptosis to produce food stasis, as is proved by the emptiness of the fasting stomach on examination, although they are undoubtedly capable of producing considerable distress and distention.

Pain may be referred to the left costal arch and is apparently due to the dragging of the gastrosplenic ligament. The condition is made worse by exercise, especially after eating, and is relieved by rest or by firm strapping of the abdomen, as by the application of a Rose's belt. Dull, diffused epigastric pain may occur after prolonged exercise and appears to be due to traction on the gastrohepatic ligament. It, too, is relieved by rest and strapping. Intermittent attacks of pain in

¹ Cincinnati Clinic, 577, December 8, 1900.

the stomach may be due to pylorospasm resulting from a chronic appendicitis, a not uncommon complication.

Pain in the back, intensified by exercise, is often of orthostatic origin and is due to faulty means of standing or walking, causing undue strain on certain ligaments.

Nausea is more frequent with gastropotosis than with simple atony, although in both conditions the characteristics of the symptom are the same, coming and going through the day without fixed relationship to the meals, or to the character of the food that is eaten. Vomiting is an uncommon symptom. When it does occur it presents nothing that is characteristic of the ailment.

Rovsing claims that hematemesis with gastropotosis is not infrequent, but with this the writer cannot agree. If hematemesis occurs the bleeding is due to some intercurrent affection rather than to the gastropotosis itself.

Auto-intoxication symptoms are present as in simple atony, and are usually more severe and continuous.

Headache, either periodical and hemicranial in type, accompanied by nausea and vomiting, or dull and occipital, is commonly observed.

2. **Symptoms of Subnutrition.**—As a rule the patients are thin and poorly nourished from infancy, and their tendency toward subnutrition is maintained throughout their life. When atonic symptoms appear loss in weight becomes more noticeable. At first the patients lose only after they have become tired and nervous and after a period of rest regain all they have lost, but when the stage of permanent atony is reached the power of recuperation is lost. When through their own volition or by injudicious medical advice the diet is unduly restricted, loss of weight often becomes extreme, so that the patients may not weigh more than 70 to 80 pounds, although for their height and bony framework a weight of 125 to 140 pounds would be normal. Every patient with gastropotosis should be regularly and systematically weighed and records kept. No treatment will be found beneficial that is not attended by a progressive gain, and the symptoms are not apt to improve until the patient is 10 to 12 pounds heavier.

3. **Symptoms of Neurasthenia.**—Symptoms of neurasthenia are regularly present. The variety of the nervous complaints is almost unlimited and their severity runs a course parallel with that of the general conditions. The danger is that we may be diverted by these nervous manifestations of disease and make the foolish diagnosis of nervous indigestion without thoroughly examining the patient and recognizing that these expressions of neurasthenia constitute but part of a broad symptom-complex which embraces also the signs and symptoms of gastro-intestinal atony and of inherent lack of assimilation.

In an article by Birtch and Inman¹ the relation between the nervous symptoms and the blood pressure is admirably considered, and conclusions are as follows:

In patients with marked abdominal relaxation the systolic blood pressure falls from 10 to 25 mm. Hg. on standing after lying. There occurs at the same time an increase in the pulse rate, undoubtedly intended to compensate for the fall in systolic pressure, but inadequate to overcome entirely the effect of gravity. If the diastolic blood pressure be well maintained the effect of gravity is compensated and circulatory disturbances are absent.

The diaphragm is an important adjunct to the circulation. Acting in opposition to the abdominal muscles it forms a respiratory pump and assists in lifting the blood from the abdominal veins to the right heart. In relaxation of the abdominal muscles there is a decrease in the intra-abdominal pressure and a decrease in the aspirating power of the diaphragm which tend to diminish the volume of blood in the arteries and to permit the stagnation of blood in the visceral veins. It is not unnatural to suppose that there must necessarily be some effect observed on the cerebral circulation to which some of the symptoms can be ascribed. In this connection Haven Emerson² writes: "Any marked loss of abdominal tone is recognized clinically as a contributing cause of venous stagnation in the abdominal viscera, and this means a delayed or insufficient return to the right heart, a diminished output and a fall in the arterial pressure, or a pressure maintained only at the expense of greater cardiac action."

Dizziness and insomnia in gastroptosis are probably the result of changes in blood pressure. According to Birtch and Inman, when dizziness is present the blood pressure shows both systolic and diastolic fall if the patient stands. The writer believes that many cases of insomnia are due to the fact that in the recumbent position the blood pressure is increased over what it is in the vertical position, producing an accelerated cerebral circulation for a considerable time after lying down, so that the onset of sleep is retarded.

The good effect of rest in bed in these cases may be due to the fact that in the horizontal position the piston-like action of the diaphragm is not required for circulation. When the vertical position is assumed the action of the diaphragm unopposed by the abdominal muscles is inadequate to do its work and maintain blood pressure as it should. The improvement noticed after applying a firm abdominal belt may be due more to the increase of abdominal pressure afforded, than to its mechanical assistance in holding the viscera in place.

¹ Jour. Amer. Med. Assoc., January 27, 1912.

² Archives Internal Medicine, June 1911, p. 754.

4. **Symptoms Due to Associated Displacements.**—The colon usually sags in its transverse portion assuming an M-shape, the splenic flexure being held well in place although the hepatic angle is lower in the abdomen than normal. With such a condition a certain degree of colon stasis is inevitable, and the absorption from its retained contents almost regularly produces the toxic symptoms that are characteristic of all forms of intestinal stasis and are classified by Arbuthnot Lane as follows:

1. The skin becomes thin, inelastic, and wrinkled. There is a general staining most marked in frictional areas, and is common in and about the eyelids. The secretions of the skin are abundant and offensive, especially in the axilla.

2. The circulation becomes enfeebled, the blood pressure lowered. Should degenerative changes later develop in the heart, bloodvessels, or kidneys, the blood pressure may be very high. The rate of the pulse is variable, being increased in frequency by any exercise, or by accumulations of gas in the abdomen. A condition closely resembling asthma may sometimes result. The extremities are cold even in the hottest weather, and the transition from the warm to the cold area is often very abrupt.

3. The temperature is habitually subnormal.

4. Muscular strength fails so that the patient is quite unable to take his daily exercise.

5. Nervous symptoms are very marked. The patients become stupid and apathetic during the day and often sleep badly at night. They awake in the morning with a headache and feel that they have derived no benefit from their sleep. Headache is a common and distressing symptom.

6. Enlargement and tenderness of the terminal joints of the hands and feet are not uncommon, and may be classed as an affective osteoarthritis due to intestinal toxins of unknown nature.

7. Lane speaks of changes in the breast which are always present when auto-intoxication has existed for any length of time, consisting in an induration in the upper and outer zone of the left breast and later in the same area on the right side. As time goes on this induration spreads, but always remains in the upper and outer segment in excess of that in the rest of the breast.

Mucous colitis almost invariably accompanies the displacement of the colon and is characterized by the passage of mucus in long, strings or in free masses more or less admixed with the stools.

In the writer's experience the subjective symptoms of such a mucous colitis are practically negligible. Pain does not seem to occur unless there be a complicating appendicitis. Unfortunately the combination

of a mucous colitis and a chronic inflammation of the appendix is not at all an unusual one.

A downward displacement of the kidney may occur, usually on the right side, occasionally on both. Displacement of the left kidney alone occurs but rarely. Fortunately the day has passed when every palpable kidney ran the risk of being sewed up in its proper place, but the idea that movable kidneys are productive of distress and discomfort to their host is dying hard. It is but rare that symptoms of any moment result from nephroptosis, no matter how extreme the displacement may be. It is conceivable that a large floating kidney may exert deleterious pressure on neighboring structures. This, however, is a clinical rarity. It is only when rotation of the kidney is allowed by the laxity of its ligaments with a resulting torsion of the ureter that characteristic symptoms appear, characterized by the symptom complex known as Dietl's crisis.

The liver may or may not be displaced downward, depending upon the strength of its supporting ligament. Hepatoptosis may, however, be quite extreme, the liver not only sagging downward but tilting forward and downward on its transverse axis so that the edge may be palpable 2 or 3 inches below the costal arch. A dragging feeling on exertion in the region of the organ is the characteristic symptom of the displacement.

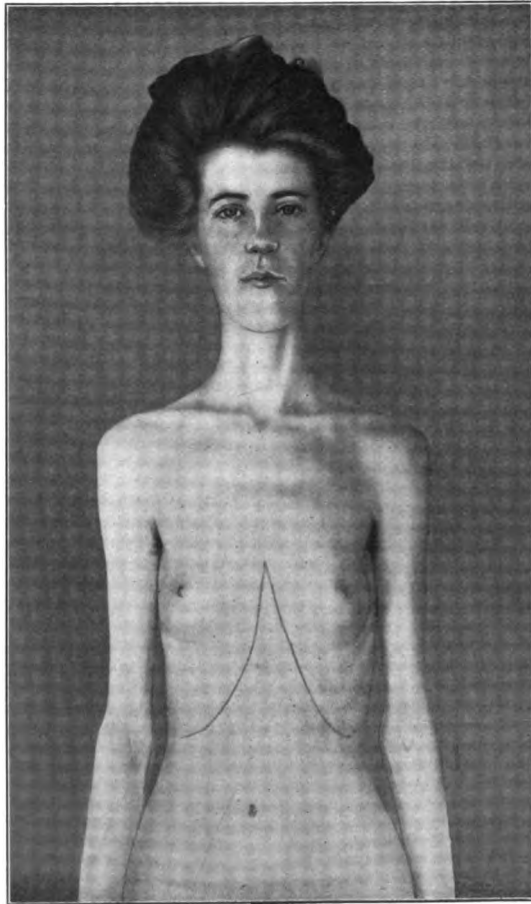
The symptoms of gastropptosis unassociated with the enteroptotic habitus, that are due to mechanical weakening of the abdominal wall, as in Landerer's cases, differ from those just described, in the absence of a definite and obvious neurasthenic state. Gastric symptoms of atony, auto-intoxication, and symptoms of intestinal origin, are usually well-marked and characteristic, but the patients do not lose in weight as in the congenital form, nor are the nervous symptoms as prominent, and although a variety of psychasthenic symptoms may occur, they do not seem to form so inherent a part in the morbid progress and are more readily amenable to treatment.

Physical Signs.—Much can be learned by the inspection of the patient. The most important presumptive evidence of the disorder is the presence of the enteroptotic habitus. The epigastric angle is abnormally sharp. The writer has made it a routine to measure the costal angle in every case, and finds that when the ribs part at a more acute angle than 50 degrees to 55 degrees the atonic and neurasthenic defects of the enteroptotic habitus are almost invariably present, even though an actual descent of the stomach may not be extreme. The distance between the ensiform cartilage is greater than the horizontal measurement from the umbilicus to the costal arch.

It has been attempted to calculate the capacity of the thoracic

abdomen and to express the result in figures. The "jugulo-pubic index" of Becker and Lenhoff is determined as follows: The measurement from the suprasternal notch to the upper border of the symphysis pubis is divided by the circumference of the waist line at its point of greatest constriction and the result is multiplied by 100. If the figure

FIG. 85



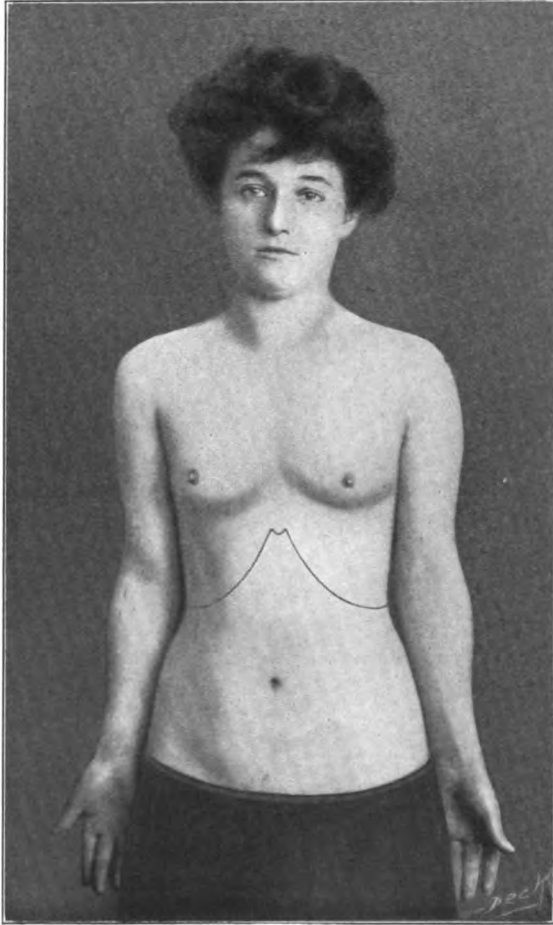
Enteroptotic habitus.

thus obtained equals or exceeds 77, the individual belongs to the enteroptotic group. In general Becker and Lenhoff's index works true, but seems to the author to possess no advantage over the simpler method of determining the epigastric angle.

The patient is slender and has but slight adipose tissue. The muscles are underdeveloped and there is a tendency on standing toward a

bulging fold of the lower portion of the abdominal wall. This, however, may not be apparent in young subjects who have not borne children. The chest is shallow, the upper ribs are far apart, the lower slanting more in a downward direction than can be observed in normal cases. These characteristics become more and more apparent during the growth

FIG. 86



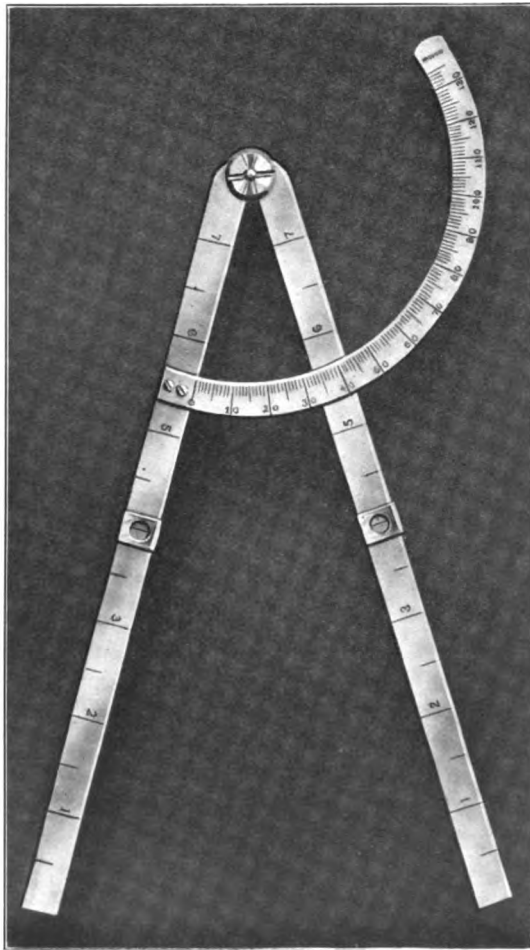
Normal habitus.

of the child to maturity. Relaxed condition of the abdominal wall should be noted and the degree of bulging of the lower abdominal zones should be observed both in the recumbent and standing positions.

Evidences of downward displacement of the stomach may be obtained with reasonable accuracy in a number of ways. The finding of the

greater curvature below the umbilicus does not necessarily prove that gastroptosis is present. Normal stomachs heavily laden with food may sag if the patient stands so that the greater curvature falls below the umbilicus. On lying down, however, the greater curvature rises 1 or 2 inches and tends to lie above the navel. All atonic stomachs are

FIG. 87

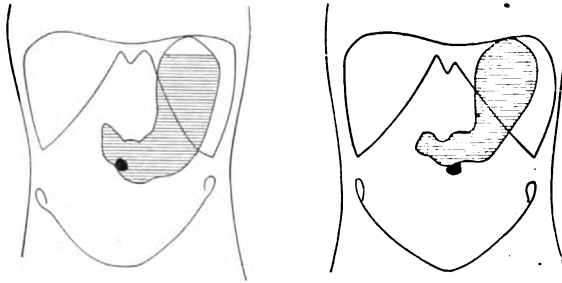


Author's angulator for measurement of the costal angle.

abnormally distensible, so that they sag downward as more and more food is put into them, but they reassume their normal position when they are free from the mechanical weight of their contents. It would, therefore, be a sad misconception to consider all stomachs gastroptotic by locating the greater curvature alone.

Gastroptosis cannot be ruled out if it be found on a single examination alone that the lower curvature in the recumbent position is above the navel, as recumbency for any length of time may allow a stomach that is ordinarily displaced to work up into normal position. To determine the size and position of the stomach three methods of examination may be employed:

FIG. 88



Normal stomach. A, in vertical position; B, in horizontal position. (Herts.)

1. The lowest point at which succussion sounds are elicited by slight palpation may be considered the lowest boundary of the organ. If the succussion sounds are not readily audible the patient may be allowed to drink a half-glass of water. The determination of the lower border of the stomach by such a method of examination is somewhat crude and often totally inaccurate.

2. Artificial dilatation is sufficiently accurate for all clinical purposes, provided that we bear in mind that all atonic stomachs, whether displaced downward or not, are abnormally distensible and that consequently overdistention will locate the lower curvature at a point to which it seldom attains under normal conditions. Inflation, therefore, should be moderate and barely sufficient to project the visible outline of the stomach upon the abdominal wall. Inflation to a greater degree than this is injurious and misleading. As a control auscultatory percussion is of service.

3. The most accurate method of all is naturally the *x*-ray examination, but it is inapplicable unfortunately as a routine. The outline and position of the stomach in health and in disease have been shown by the *x*-ray to be different from what we were formerly led to suppose.

Splashing on light percussion are more easily elicited in gastroptosis than in simple atony, because a greater portion of the stomach comes into direct contact with the abdominal wall. The diagnostic significance

PLATE XIV

Fig. 1



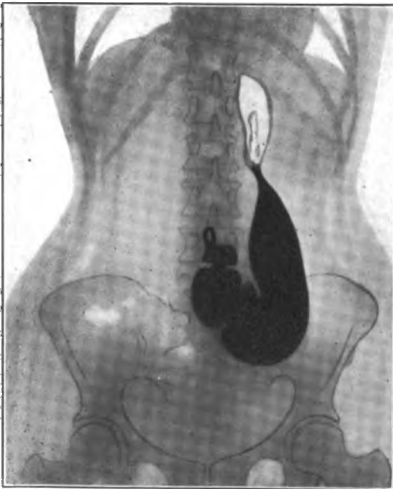
Fig. 2



Fig. 1.—Vertically Placed Stomach of a Child, Twelve Years of Age, with the Enteroptotic Habitus. (Radiologist, Dr. Leaming.)

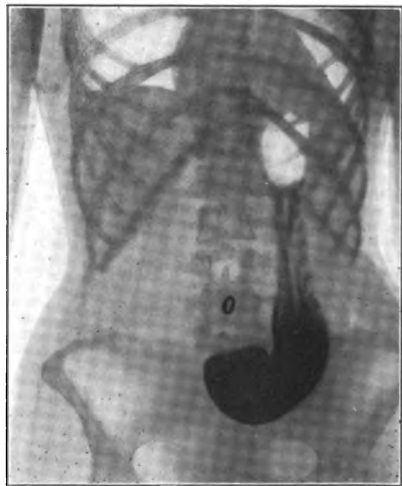
Fig. 2.—Gastropnoxis without Atony. (Radiologist, Dr. Le Wald.)

Fig. 8



Gastropnoxis with Moderate Atony. (Radiologist, Dr. Leaming.)

Fig. 4



Gastropnoxis with Marked Degree of Atony. (Radiologist, Dr. Leaming.)

of succussion has been fully described under the heading of Atony, page 319.

No evidence of increased peristalsis can be obtained in uncomplicated gastroptosis, and the absence of these physical signs seem somewhat to disprove the idea that in gastroptosis mesenteric constriction or duodenal kinks produce a definite obstruction to the onward passage of the stomach contents. A certain peristaltic unrest may occasionally be observed in thin subjects with greatly relaxed abdominal wall and is distinguished from an increased peristalsis by the small size of the waves that rise and fall, and by the absence of the stately march from left to right.

The abdominal aorta is usually felt pulsating forcibly in the epigastrium, although no lateral expansion is observed as in aneurysm. A systolic murmur is often audible, which may be transmitted downward and heard in the femoral vessels. Tenderness over the aorta is usually quite marked, especially when pressure is made over the celiac plexus just above the umbilicus and a little to the left of the median line.

Corroborative evidence is afforded by demonstrable ptoses of other organs, especially the kidney and colon. The lower pole of the right kidney is normally palpable in the majority of women, and it is only when the greater part of the organ is palpable that an actual pathological nephroptosis is present. Colo-ptosis may be demonstrated by the inflation of the colon by air or by locating, after the injection of water, succussion sounds below the lower curvature of the stomach that has previously been located by other means.

For this test to be of any service an examination of the colon should be made when the stomach is empty, so that no confusion may be caused by the presence of succussion sounds in the colon and stomach at the same time. The most certain test is by the x-rays.

Diagnosis.—Gastric Analysis.—Fasting Stomach.—The fasting stomach in gastroptosis should regularly be empty. Food-stasis and fasting hypersecretions are not observed unless complications be present. In 200 cases of the disorder in which examination of the fasting stomach was made in gastroptosis there were found in the fasting stomach:

0 to 25 c.c. of fluid in	173 cases
25 to 50 c.c. of fluid in	19 cases
50 to 75 c.c. of fluid in	4 cases
75 to 100 c.c. of fluid in	3 cases
100 to 125 c.c. of fluid in	1 case

In over one-half of the cases in which an amount of fluid over 50 c.c. was obtained the existence of chronic appendicitis could be demon-

strated, and in a number of these from whom the appendix was removed, the hypersecretion was not observed after the operation, indicating that it took its origin from pylorospasm, depending upon the appendix rather than due to the gastroptosis itself. That the slight amount of hypersecretion observed in some of the cases was not due to the gastroptosis itself seems to be proved by the writer's analysis of 100 cases of gastroptosis divided into 3 groups of slight, moderate, and marked degrees of atony.

EXAMINATION OF FASTING STOMACH IN GASTROPTOSIS WITH VARYING DEGREES OF ATONY

1. Slight atony, 38 cases	$\left\{ \begin{array}{l} 0 \text{ to } 25 \text{ c.c., } 36 \text{ cases} \\ 25 \text{ to } 50 \text{ c.c., } 1 \text{ case} \\ 50 \text{ to } 75 \text{ c.c., } 1 \text{ case} \end{array} \right.$
2. Moderate atony, 44 cases	$\left\{ \begin{array}{l} 0 \text{ to } 25 \text{ c.c., } 38 \text{ cases} \\ 25 \text{ to } 50 \text{ c.c., } 4 \text{ cases} \\ 50 \text{ to } 75 \text{ c.c., } 2 \text{ cases} \end{array} \right.$
3. Marked atony, 39 cases	$\left\{ \begin{array}{l} 0 \text{ to } 25 \text{ c.c., } 37 \text{ cases} \\ 25 \text{ to } 50 \text{ c.c., } 2 \text{ cases} \\ 50 \text{ to } 75 \text{ c.c., } 0 \text{ cases} \end{array} \right.$

It would, therefore, seem quite conclusive that atony, which is the primary essential of gastroptosis, does not influence in the least the conditions of the fasting stomach, nor does it appear that obstructive lesions ascribed to the descent of the stomach and intestines, such as mesenteric traction and duodenal kinks, are sufficiently obstructive, if obstructive at all, to alter the condition of the fasting state.

Test Breakfast.—Test breakfast, as a rule, is well digested, of homogeneous consistency, and without demonstrable signs of gastric catarrh. The general appearance of the contents is that of atony, often slightly more abundant than normal, and settling on standing into two layers, the liquid stratum not being more than equal the depth of the sedimentary deposits of breadstuffs. The acidity is normal or slightly acid in over four-fifths of the cases, as is shown by the result of the writer's examinations in 250 cases.

Achylia was present in	9.5 per cent.
Subacidity was present in	8.0 per cent.
Normal acidity was present in	55.6 per cent.
Hyperacidity was present in	27.4 per cent.

The acidity in gastroptosis depends largely upon the degree of the associated atony, the more marked the atony the greater is the tendency toward hyperacidity. In the writer's series:

In gastroptosis with subacidity, atony was marked in $\frac{1}{3}$ of the cases
In gastroptosis with normal acidity, atony was marked in $\frac{1}{3}$ of the cases
In gastroptosis with hyperacidity, atony was marked in $\frac{2}{3}$ of the cases

The proportion of cases complicated by achylia, 9.5 per cent. may at first sight seem rather large, and may suggest that gastroptosis predisposes toward such a reduction of gastric acidity. Achylia in the writer's experience occurs, however, in nearly 7 per cent. of all patients applying for relief of gastro-intestinal disorders. According to these figures achylia would naturally complicate 7 per cent. of the gastroptosis cases. As a matter of fact it complicates but 9.5 per cent., the difference being so slight as to make a causal relationship between the two diseases highly improbable.

Prognosis.—The prognosis is good for the prolongation of life and good for a reasonable degree of well-being to enable the patient to live his allotted life with comparative comfort. Much depends, however, upon the ability of the patient to live within physical and nervous limitations. Enteroptotic individuals who are congenitally neurasthenic can never hope to be robust and of good endurance, but they will suffer from their digestion whenever they run down from any cause whatever. The prognosis in such cases naturally depends upon the ability of the patient so to adapt his life that his meagre nervous and physical resources are conserved rather than wasted. Under proper hygiene and treatment results are often obtained that are satisfactory to the physician and patient alike, and occasionally the result is both brilliant and lasting.

Treatment.—Prophylactic Treatment.—It must be recognized that gastroptosis may exist without symptoms until the stomach becomes atonic. The prophylactic treatment is therefore directed toward the prevention of atony. Those individuals with the enteroptotic habitus are by nature unable to stand the storm and stress of life, and therefore not only should they be spared as far as possible excessive physical strains and long strenuous hours of work, but by outdoor life and ample amounts of rest and sleep they are to be built up and rendered hardy and more resistant to the demands of daily life. Especially is this prophylactic treatment indicated in enteroptotic children during their years of rapid growth and development. Excessive water-drinking at meals should be prohibited. The daily work should not be resumed after acute illness until the full strength has returned. After childbirth, rest in bed should be enforced for a much longer period of time than in normal individuals, so as to allow the abdominal walls to regain their tone, and upon convalescence a well-fitting abdominal belt should be worn for at least six months.

Medical Treatment.—The medical treatment is devised to meet three indications:

1. To conserve the muscular lack of tone and overdistention of the atonic stomach by diet, rest, and abdominal support.
2. To produce a gain in weight.
3. To counteract the neurasthenic state.

Either an ambulant or a rest cure treatment may be indicated.

Ambulant Treatment.—If the symptoms are not severe and not attended by marked loss of flesh and strength, it may be possible to effect a cure while the patient is up and around. The ambulant treatment may also be tried when the circumstances of the case or the inclinations of the patient render a rest cure impossible or inexpedient for the time being. It is well, however, to explain to the patient that such a treatment is but experimental and that a rest cure regime may become necessary in case of failure.

Dietetic Treatment.—The diet is that of atony. The main indication is to reduce the mechanical bulk taken at any one meal and to divide the food into small quantities taken at more frequent intervals. Liquids should be taken sparingly at the meals although they may be taken in the times between, in doses sufficient to assuage thirst, provided that more than one-half glass is not taken at any one time. The diet should be varied and individual tastes should be consulted as far as possible, as the tendency always is for these patients to eat too little rather than to eat too much. Too much attention should not be paid to foolish whims and fancies which result in the cutting down of the diet to a starvation-point. An increase in fats is usually desirable. Fresh butter and cream are to be advised, or Russell's emulsion may be given. As the digestive power of the stomach is usually good, the quality of food may be varied, but should achylia exist, red meats should be reduced, with a corresponding increase in the carbohydrates and fats. A simple diet for gastroptosis is as follows:

BREAKFAST.—*Allowed:* Cup of coffee or cocoa, with cream and sugar. Saccharin may be used instead of sugar if preferred. Cereal with cream and sugar. Two soft-boiled or poached eggs or minced chicken. Rolls, toast, pulled bread, or zwiebach. Maximum amount of butter, preferably unsalted. Strained honey, such as Sheffield Farms. Orange marmalade or any Dundee jam.

None: No hot bread; no fruit.

10 to 11 A.M.—*Choice of:* Glass of top milk or cream, or milk and cream, and crackers. Cup custard; junket; egg shake or raw eggs; chicken sandwich; malted milk; buttermilk or lactone milk; Russell's emulsion.

LUNCHEON.—*None:* No soup. Liquids restricted to less than one glass. No steak, roast beef, pork, salt fish, or shell-fish. No sweet-breads or kidneys. No radishes, raw celery, or anchovy.

Allowed: Caviar, olives.

Choice of: Fresh fish, chicken, lamb, mutton, simply prepared ragout; oysters in any form; fowl of all kinds except domestic duck or goose; lean broiled or boiled ham.

Freely: Peas, beans, spinach, samp, rice, macaroni, spaghetti, with cheese if preferred. Any vegetable that may be put through a purée sieve, such as turnips, carrots, etc. Potatoes may be taken in any form but fried; boiled potatoes to be taken very occasionally and then thoroughly masticated. Boiled onions, tender beets, and oyster plant.

Occasionally: Cauliflower, Brussels sprouts, stewed celery, artichoke, asparagus tips. Salad with French dressing.

DESSERT.—*Choice of:* Rice pudding, farina, corn-starch, blanc-mange, prune soufflé, tapioca; ice-cream, but no fruit ices; baked apple with cream; stewed figs and prunes.

Occasionally: Simple cake, such as pound cake, sponge cake, etc.

None: No oranges or grape fruit or raw apples.

Allowed: Cream cheese, Roquefort, Camembert, Brie, Neufchatel, or pot cheese.

4 P.M.—Same variety as 11 A.M., with the additional choice of cocoa or chocolate with cream and sugar; or a farinaceous dessert, such as on luncheon list.

DINNER.—Same variety as for luncheon.

While liquids are restricted at meals, water may be freely taken between meals.

Drug Treatment.—During the early part of the treatment patients are usually keyed up and on high tension, and the writer has found it desirable in almost every case to reduce this excitable nervous state by suitable medication.

Of all forms of soothing medication, the following prescription has proved the most serviceable, and the writer employs it almost as a routine measure in his cases.

R—Resorcin resublimat. (Merck)	3j
Chloral hydrat.	3ss
Strontii bromid.	3iiss
Aq. chloroform.	3iv
Spirits anise	gtt. viij

M. Sig.—Teaspoonful in a wineglass of water three times a day after eating.

After about a week a change should be made to a more stimulating form of treatment, of which nux vomica or physostigma is the

essential feature. The following prescriptions have been found serviceable:

- R—Tinct. physostigma,
 Tinct. nucis vomicæ āā 3iiss
 Sodii glycerophosphate 3v
 Glycerole diazyme (Fairchild) 3iv
 Aquæ ad 3viiij
- M. Sig.—Dessertspoonful in a little water three times a day after eating.
- R—Tinct. nucis vomicæ 3v
 Elxir calisaya ad 3iv
- M. Sig.—Teaspoonful in water after eating three times a day.
- R—Eserine gr. ʒss
 Ferri et quin. citrat. gr. iij
 Calcii glycerophosphate gr. viij
- M. ft. caps. no. j. Dentur tales doses, no. xxxiv.
- Sig.—One capsule three times a day after eating.

It is unwise, however, to make the change of treatment too suddenly. The writer recommends that for three days one of the nux vomica preparations be given after breakfast and the bromide mixture after lunch and dinner; that for the next three days the nux vomica be given after breakfast and lunch, the bromide after dinner alone, and for the next three days, three doses of the nux vomica preparation be given after meals, while the bromide is to be given at bedtime. This latter combination may be continued for at least two months. During the early part of the treatment the bowels should be opened, preferably by a saline having a cholagogue effect. The following prescription has been found serviceable:

- R—Sodii benzoat. 3j
 Sodii salicylat. 3ij
 Sodii sulphat. 3vj
 Magnesii sulphat. 3xj
 Tinct. nucis vomicæ 3ss
 Aquæ 3viiij
- M. Sig.—Tablespoonful in three-fourths of a glass of water on rising.

Effervescing sodium phosphate may be advised. The dose of the aperient should be so regulated so that no more than one liquid or two semiformed movements result. Should auto-intoxication symptoms be prominent, intestinal irrigations two or three times a week may be useful. The technique of the proper form of irrigation is given under Atony on page 331.

After the bowels have been regulated by the saline, laxative articles of diet should be added and the dose of the aperient gradually reduced. Stewed fruit at or after the meals (never before) or one-half a wine-

glassful of olive oil or paraffin oil at bedtime, or the increase of sugar, especially of lactose, may be found serviceable. The more sugar of milk can be worked into the diet the better. Coarse breads and the use of bran in biscuits or in the form of bran cakes are serviceable

FIG. 89



Author's belt for enteroptosis. The uplift is to be especially noted.

in increasing peristalsis. Peristaltic hormone may be given either by mouth or by intermuscular injection, and either is noticeably efficacious or absolutely disappointing.

Hygienic Treatment.—Patients should not exercise if they can help it directly after eating. Two hours' rest in bed during the day is always

advisable, and is generally possible if the patient so wills it to be. The best hours for rest are directly after lunch, but in the short afternoons of winter the rest may be taken before dinner. A tight-fitting abdominal belt, or a corset that exerts a belt-like pressure on the lower abdomen, leaving ample space above for deep respiration, is of service in almost

FIG. 90



Ordinary abdominal belt, not to be advised as it has no uplifting effect.

all the cases, not only because of the support which it gives to pro-lapsed viscera, but because counter-pressure upon the abdomen prevents, to some extent at least, the baneful effects of gravity in producing a lowered blood pressure when the patient stands. The physician should personally see to it that the belt is well-fitting and serves its purpose. The majority of belts in the market are practically broad elastic belts

about the hips, pressing backward upon the abdominal wall without any traction upward. The belt used by the writer is so constructed that it lies low in front and rises high in the back, so that the pressure is not only backward but upward. Perineal straps seem to be a necessity, as otherwise the belt rides up in front and exerts no upward traction.

FIG. 91



Good straight front corset. To be recommended.¹

The belt should be adjusted before the patient rises in the morning, so as to retain the organs as far as possible in the position which they assume during recumbency, and the belt should be worn continuously

¹ This corset is made by Mme. Rosso, 27 W. 36th St., New York City.

during the day. If the belt be well-fitting the use of objectionable hernia pads may be avoided.

A variety of corsets may be employed, some of the simple straight front variety and others with an inner belt of elastic webbing.

FIG. 92



Combination corset, showing inner elastic belt attached to the side of a straight front corset.

Mechanical support of the stomach by adhesive strapping is of service for short periods of time, but naturally cannot be long continued. The best method of adhesive strapping must be credited to Dr. Achilles Rose.

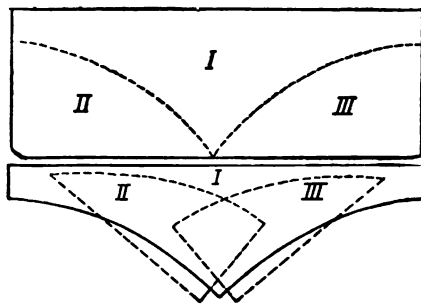
For the application of a Rose belt a yard strip 7 inches wide of ZO adhesive moleskin plaster is to be used, and cut in three pieces, as shown in Fig. 94. The piece marked *I* is first to be applied, the apex

FIG. 93



Combination corset. The straight front corset is fastened. The minor elastic belt shown in the preceding photograph is fastened at the side by the tape.¹

FIG. 94



Rose's belt.

¹ This belt is made by Mme. Rosso, 27 West 36th St., New York City.

lying just above the symphysis, the ends passing upward and overlapping in the small of the back. The pieces *II* and *III* are then to be applied as shown in Fig. 95.

A simple method for mechanical treatment is advised by McCaskey.¹ The hair on the pubes having been shaved, a strip of zinc oxide adhesive

FIG. 95



Roe's belt as applied.

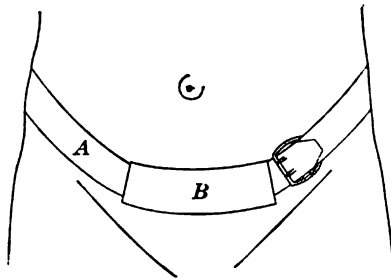
plaster, 2 or 2½ inches wide and 5 or 6 inches long, is placed transversely across the extreme lower abdomen as near as possible to the pubes. To each end of this strap is attached a bandage of about the same width, long enough to pass around the body above the iliac crest, and there be tied or otherwise fastened. If the ends of the plaster become

¹ Jour. Amer. Med. Assoc., October 28, 1911.

loosened and pulls up by traction of the bandage, narrow vertical strips of plaster may be applied across each end of the adhesive strap and applied to the skin above and below. The bandage is well padded with cotton so as to prevent irritation of the skin beneath it and to permit of its being drawn as tightly as possible to furnish the necessary support from below.

Under the ambulant treatment just described, a gain in weight is the main indication that the treatment is beneficial. If the patient does not gain within six weeks there is very little use in going on with the case while the patient is up and about, but as long as the gain continues the treatment may be persisted in. Some patients do not gain at all, others gain for a time and then relapse, so that at the end of three months they are no better than when they started. For these patients nothing but a rest cure will be of service.

FIG. 96



A device for the mechanical treatment of Glenard's disease. A, double-padded bandage; B, zinc oxide strip. (McCaskey.)

Rest Cure Treatment.—A rest cure treatment with forced feeding is indicated in all cases with aggravated symptoms attended by an extreme loss of flesh and strength and in all other cases in which the ambulant treatment has not been followed by a progressive gain in weight. Many patients suffer from the mechanical overdistention of the stomach by food when they are sitting or standing, and consequently cannot eat enough with comfort to gain in flesh and strength, but they can eat a sufficient quantity while they are in bed. In other cases nervous symptoms induced by a lowered blood pressure on standing are controlled by prolonged recumbency. The patient should be kept in bed for at least four weeks, rarely over that time. The services of a tactful and congenial nurse are almost essential for good results of the treatment. Semi-isolation should be insisted upon. Congenial, cheerful friends are usually better than oversympathetic relatives, but as it is difficult to draw the line, the fewer that see the patient the better. The patient may go to the toilet or sit up while the room is prepared,

but at other times should remain in bed. Except at meals more than two pillows should not be allowed.

During the first two weeks hot applications should be applied to the abdomen, either in the form of the Priesnitz umschlag or the electric pad, under which should be placed one layer of damp flannel. The monotony of the afternoon may be broken by a hot drip sheet for a half-hour followed by a cold spinal sponge and an alcohol rub.

The diet and the medication are the same as in the ambulant form. Some of the patients, especially those who have been unable hitherto to take sufficient nourishment without discomfort, may complain of the increased quantity of food which they are obliged to take, and it may be that they are more uncomfortable under the full diet than they were before the treatment began. Under these circumstances it is well to cut down the quantities but to continue with the varied quality of the meals indicated in the diet list. After about a week the quantity can be gradually increased.

Intragastric faradization may be tried in those who are peculiarly susceptible to the good effects of suggestive treatment. It serves to relieve monotony and to exercise the abdominal wall, but its actual value in curing atony is very problematical. A scheme for the day that has been found by the writer to be practical is as follows:

- 7.45 A.M. Glass of water or saline draft. Wash hands and face, clean teeth, change hot applications.
- 8.15 A.M. Breakfast.
- 8.30 A.M. Rest.
- 9.30 A.M. Stroking massage to the abdomen.
- 9.45 A.M. Intestinal irrigation.
- 10.45 A.M. Warm pad; cold spinal douche; make the bed.
- 11.00 A.M. Nourishment.
- 11.00 A.M. Rest.
- 1.00 P.M. Lunch.
- 2.00 P.M. Rest alone; nurse goes out.
- 4.00 P.M. Abdominal massage, stroking or circling movements, or intragastric faradization.
- 4.30 P.M. Nourishment.
- 6.00 P.M. Drip sheet; cold spinal sponge; alcohol rub.
- 7.00 P.M. Dinner.
- 8.15 P.M. General massage.
- 9.15 P.M. Wash hands and face; make the bed for the night.
- 9.30 P.M. Nourishment; clean teeth.
- 9.45 P.M. Lights out.

Hot applications changed every two hours in the day and left on all night.

The first sign of improvement by such a treatment is a gain in weight, and the weight curve follows one or two types. Gain in weight may be progressive from the start, averaging frequently one-half pound a day, occasionally dropping a little, and usually remaining stationary during the menstrual period. When the patient reassumes family life at the termination of the rest cure, there is often a fall in weight and a return of the old symptoms; but the relapse is temporary, and is succeeded by a further progressive gain. In other cases it may be almost impossible during the rest cure for the patient to gain much weight, so that at the end of the four weeks he may be but 2 or 3 pounds heavier than at the start. The temporary loss of weight usually occurs at the end of the treatment, so that the patient loses what he has gained, but succeeding this, there is a progressive gain which may ultimately be as great as that observed in the first set of cases.

The patients should always be warned of the probable return of their symptoms during the week following the termination of the rest cure.

About every ten days during the period of forced feeding patients complain of "feeling bilious." The tongue is coated, the breath offensive, and the appetite fails. These symptoms are readily controlled by small doses of calomel, or by the following prescription:

R—Massæ hydrarg.,
 Ext. leptandra,
 Bile salts (Fairchild) āā gr. ½
 M. ft. caps. no. j.
 Sig.—One three times a day for three days.

Succeeding the rest cure the patients should follow the rules of management laid down in the ambulant form of treatment and should be under observation for at least six months. Systematic weight records should be made and the causes for any loss should be ascertained.

Surgical Treatment.—To the surgeon the displacement of the stomach is a matter of prime importance, and there are those who advocate surgical measures to restore the stomach to its proper place and at the same time to correct in some instances the results of duodenal or intestinal kinks and stasis by appropriate means. Beyea was among the first in this country to perform the operation of suturing the gastro-hepatic omentum to secure elevation of the stomach, and has treated 26 cases in this way, all of which had resisted skilled medical treatment. Beyea takes the conservative stand that the operation should be done only when other treatment falls short of a cure. Rovsing¹ is a more enthusiastic advocate of the operation, having an experience of over

¹ Jour. Amer. Med. Assoc., lix, No. 5, p. 334.

163 cases in which gastropexy was done. In his series hepatopepy was done in 68, and in 4 cases it was necessary to resect part of the left lobe of the liver in order to gain access to the gastrohepatic omentum. In 10 of the cases secondary unilateral or bilateral nephropexy was necessary before an absolute result could be obtained.

Rovsing anchors the upper line of the stomach by leading strong silk sutures on the serous coat of the stomach parallel with the lesser curvature through the anterior abdominal wall, so that the stomach lies flat against the abdominal parietes without shrinkage or folding, thus obtaining a perfectly secure and solid adhesion. Rovsing has collected the reports of 93 additional operations performed by Scandinavian surgeons according to his technique, and adding these results to the 163 cases of his own has tabulated the end results in the 256 cases as follows:

Cure	63.2 per cent.
Great improvement	12.8 per cent.
Improvement	7.0 per cent.
Slight improvement or no change	12.8 per cent.
Deaths	4.6 per cent.

Gastrojejunostomy formerly in vogue in the hands of a few surgeons in the treatment of gastroptosis has been entirely abandoned for the reason that in this condition pyloric or duodenal obstruction does not occur, and there is, therefore, no reason for performing the operation. Of late surgery has grown more radical in the treatment of these cases. Not only is gastropexy advised, but partial gastrectomy with anastomosis of the duodenum to the stomach, resection of the colon in whole or in part, or ileocolostomy are being recommended. To this radical surgery the writer takes exception. An experience of a little over 600 cases in private practice in which the disease was treated medically, seems to him to indicate that the end-results obtained are immeasurably better than those recorded by Rovsing in his surgical patients. It is not the displacement of the stomach that produces the symptoms, it is the atony to which it is predisposed, and the atony can be more readily treated by medical than by surgical means. Unless the mortality rate can be reduced from Rovsing's figures, surgery is not lightly to be advised.

Any operation performed on a patient with visceral ptoses and the enteroptotic habitus is apt to change a latent neurasthenic into an active one.

CHAPTER XVII

HYPERACIDITY

HYPERACIDITY may be defined as an abnormal increase in hydrochloric acid during the digesting period, the phenomenon disappearing as soon as the stomach empties itself. The term is unsatisfactory because it indicates only a secondary disturbance of the stomach which is common to a great variety of disorders both organic and functional, acute and chronic. It is as indistinctive as "fever" or "bronchial breathing." Cases are commonly designated as hyperacidity or hyperchlorhydria that are really examples of ulcer, cancer, muscular insufficiency, or gall-bladder or appendicular disease without any attempt at a more accurate classification.

Hyperacidity is to be sharply differentiated from hypersecretion. In hyperacidity the percentage of acid is increased without increase in the quantity of fluid secreted, while in hypersecretion the whole quantity of gastric juice is increased over and above that required for the purposes of digestion, either during the digesting period alone (alimentary secretion) or poured out as well at times when the stomach should be empty (continuous hypersecretion). The two conditions are often combined; the majority of hypersecretions are hyperacid, but on the other hand hyperacidity exists frequently enough without any hypersecretion at all. Again we must sharply differentiate between those cases of hyperacidity that are due to recognized forms of organic disease (secondary hyperacidity) from those instances in which no definite cause for the hyperacidity can be ascribed, and which we consider by exclusion to be of functional origin (primary hyperacidity). Many glaring errors of diagnosis are committed by classifying together a variety of diseases having only this one symptom of hyperchlorhydria. Among the organic causes which may give rise to secondary hyperacidity may be numerated:

1. Gastric and duodenal ulcer.
2. Gastric cancer.
3. Hyperacid gastritis.
4. Pyloric stenosis—benign, malignant or spasmodic.
5. Irritative lesions of the gall-bladder or appendix. In addition to these organic lesions we must add
6. Gastric myasthenia or atony—either with or without gastropptosis.

In 200 consecutive cases in which the total acidity of the gastric contents after the Ewald test breakfast was 70 or over, irrespective of whether hypersecretion was present or not, the following percentages were found for the various causes of the condition.

Gastroptosis	20.5 per cent.
Ulcer	19.0 per cent.
Chronic appendicitis	11.5 per cent.
Atony	11.0 per cent.
Diseases of the gall-bladder	5.5 per cent.
Chronic acid gastritis	5.5 per cent.
Cancer	4.0 per cent.
Hypersecretion of unknown origin	2.0 per cent.
Benign pyloric stenosis	1.0 per cent.
Unknown or functional	20.0 per cent.

Of these cases 21 per cent. were accompanied by hypersecretion while in 79 per cent. gastric juice was not secreted in abnormal quantity either in the fasting or in the digesting state. It is evident that to call all these cases hyperacidity indicates lack of knowledge and of careful efforts of diagnosis that are quite inexcusable, and yet this is what is happening every day. After organic causes are excluded there remain a number of instances in which no definite lesion can be discovered and in which the cause for the hyperacidity remains in doubt. In our present state of knowledge we are forced to class these cases as of functional origin provided that we do not regard the use of the term as final but keep our mind open and our senses alert to make a better diagnosis in time. *The diagnosis of hyperacidity must always be a provisional one.*

Frequency.—The frequency of hyperacidity is difficult to determine with any accuracy. The general consensus of opinion is that about one-half the dyspeptics who apply for treatment suffer from this disorder. In Germany, Jaworski found hyperacidity in 75 per cent. of all patients examined. According to this writer it is most commonly encountered in Polish Jews. In New York, Einhorn reports 50 per cent. of all patients examined by him to be thus affected. Friedenwald, in Baltimore, found hyperacidity present in 63 per cent. of 2000 private patients examined. In France, Matthieu and Rémond report the frequency as 30 per cent., Bouveret as 25 per cent. On the other hand, Fenwick, in England claims that but 4.8 per cent. of his hospital cases and 9.2 per cent. of his private patients show hyperacidity. In the writer's experience 17.4 per cent. of private patients with indigestion show superacid gastric conditions including both hyperacidity and hypersecretion, but that only 13.8 per cent show hyperacidity without actual increase in the amount of gastric juice secreted. His experience

at Bellevue Hospital leads him to the conclusion that hyperacidity is about one-half as frequent in hospital as in private cases. There are several reasons for these differences of opinion as to the relative frequency of the disorder.

There is unfortunately no fixed standard of gastric acidity that is to be regarded as normal. Some observers diagnosticate hyperacidity whenever the total acid is over 50, others only when the acidity runs to 60 or 70, while many writers do not give any indication at all as to what they regard as the dividing line between acidity that is normal and acidity that is excessive, so that we do not know what they really mean by hyperacidity. The writer believes that in private patients coming from New York and the adjacent cities, the normal limit of gastric acidity ranges from 50 to 70, and that it is only when the total acidity runs beyond this point that it may be regarded as abnormal. In hospital practice the normal acidity is somewhat lower, ranging from 50 to 60. These figures apply only to the analyses of the Ewald test breakfast. For ordinary meals and test dinners which include meat, at least 10 points must be added.

There are undoubtedly variations in gastric acidity in different places and among different races, due not alone to temperamental and racial peculiarities but also to environment, to variations in diet and to the varying frequency of organic diseases of the stomach, such as acid gastritis or ulcer. It may be assumed that hyperacidity is most common in Germany and the Northern European countries, less so in France, while in England and the United States the disorder is comparatively infrequent.

In many medical essays on this subject there are included cases of hypersecretion, ulcer, cancer, of reflex pylorospasm from gall-bladder or appendicular diseases, together with cases of hyperacidity that are due to gastroparesis and to atony.

While all these pathological conditions show hyperacidity in common, it is obviously an error to group them together.

The only proof of hyperacidity is the analysis of the gastric contents. We cannot make a diagnosis on the history alone, as the feeling of heartburn may accompany subacidity or even achylia, while, on the other hand, we meet with extreme degrees of hyperacidity which give rise to no gastric discomfort whatever. In spite of this fact patients are often said to be suffering from hyperacidity, without proof of the diagnosis by gastric analysis. We have no means of determining the relative number of individuals who have hyperacidity without symptoms, and who, therefore, never have reason to ask medical advice for indigestion. It is probable that the majority of patients who have hyperacidity remain unexamined and untreated.

Etiology.—Dietetic Errors.—Dietetic errors are generally supposed to be the most prolific cause for the hyperacid state. Overexcitability of the gastric glands is commonly attributed to the character of the food that is eaten. Overindulgence in condiments, spices, coffee, alcoholic beverages, sweets, and richly prepared food are cited as common causes for the ailment, while the administration of certain drugs, such as gentian, capsicum, nux vomica, and the essential oils, oil of copaiba and sandalwood, may be followed by attacks of the disorder. Insufficient mastication and the bolting of food in large masses, food insufficiently softened by cooking, coarse bread, and an excess of hard vegetables are said to be exciting causes for the complaint.

Food that is rich in nitrogenous elements is known to produce a more acid gastric juice than is a diet of carbohydrates. As long as the supply of food continues, this overacidity continues without ill effects, but should the diet be suddenly restricted from any cause, the excessive acidity no longer neutralized or combined with food, may make itself felt by heart-burn and other symptoms of indigestion. Fenwick lays stress on this point and claims it is for this reason that so many bon vivants suffer from acid dyspepsia, when owing to an attack of gout or other disease they are obliged to confine themselves to a limited amount of food. Sooner or later the stomach adapts itself to the requirements of a new diet. Pawlow found that whenever in feeding animals the kind of food is altered and the new diet maintained for a length of time, the digesting quality of the juice becomes day by day more and more adapted to the new dietetic regime.

When we come to consider the influence which food has upon gastric secretions, in the light of recent scientific investigation we are forced to admit that it has been very greatly overestimated. It has been definitely determined by Pawlow, Hertz, and others that the mucous membrane of the stomach is totally insensitive to sensory stimulation and that the direct contact of the interior of the stomach with irritating food, drugs, acids, foreign bodies, and other forms of stimulation does not in the least influence the forms of gastric juice. "The mechanical stimulation of the stomach wall by food thus causing further the secretory work of the glands is nothing less than a sad misconception." (Pawlow.)

Hyperacidity is more commonly seen in private practice when the food is well cooked and wholesome than it is in hospital practice in which the patients habitually indulge in dietetic errors of the grossest sort. The writer's experience on this point coincides with that of Fenwick. Among his patients with hyperacidity the writer cannot convince himself that the various dietetic indiscretions so commonly causative of a hyperacid condition are any more frequent or flagrant

than can be observed among a similar number of patients whose digestions are without flaw or blemish. It is well upon general principles to insist upon the importance of simple and wholesome food, and upon its thorough and leisurely mastication; but the writer believes that more harm than good is done by overdieting the patients and by placing them on a dietary that is insufficient and unappetizing.

Motor Errors.—Motor errors both small and great are probably the commonest form for hyperacidity, and the more carefully we study our cases the greater their importance grows upon us. On glancing at the table, page 460, of the various causes found for hyperacidity the number in which motor inadequacy was a prominent factor is surprisingly great: 20.5 per cent. occurred with gastropnoia and atony, 11 per cent. with uncomplicated atony, making 31.5 per cent. of all cases depending upon atonic error. Ulcer was present in 19 per cent. It is generally accepted that in ulcer the acidity increases the nearer is the ulcer to the pylorus, the reason being that in ulcers at or near the orifice a certain degree of stenosis is present, either structural or spasmodic, the effect of which is regularly to cause a food retention that leads to hypersecretion and hyperacidity. Saddle-back ulcer of the lesser curvature may also interfere with gastric motility and be followed by the same secretory excess. In the 4 per cent. of hyperacidities accompanying cancer the site of the growth was pyloric in every instance, while in the hyperacidity that occurred with chronic acid gastritis evidences of slight motor error were found in all the cases.

Diseases of the gall-bladder and appendix are often complicated by hyperacidity resulting from pylorospasm. Certainly motor error of such a nature exists in a large proportion of cases of hyperacidity accompanying cholecystitis or chronic appendicitis. Of two hundred cases of cholecystitis in the writer's private practice, hyperacidity was present in 30 per cent. When, therefore, we sum up the cases of hyperacidity from whatever cause it may arise, motor error was present in 75 per cent. of the writer's cases. This is a higher estimate than is generally conceded.

Graull¹ found hyperacidity in 50 per cent. of his cases of atony, while Kaufmann² makes a somewhat higher estimate. As a clinical fact, whenever food exit is delayed, hyperacidity appears, and the more careful is our examination of patients with hyperacidity the larger is the number of gastric atonies and motor errors of insufficiency that are discovered. A further description of the relationship between motor errors and gastric secretion will be found in the article on Hypersecretion.

¹ *Archiv für Verdauungskrank.* xiii, 627.

² *Zeitschrift klin. Med.*, 1905, lviii, 491.

Oswald reports that among his cases of chlorosis hyperacidity was present in 85 per cent., while Friedenwald has found hyperacidity in 75 per cent. The writer cannot place his figures nearly as high as these, but believes that hyperacidity does not occur with chlorosis except in those chloranemic patients who have concomitant gastric atony and in whom hyperacidity exists as the result of atony rather than of the anemia.

Constipation.—The correlation between constipation and hyperacidity has not been particularly noticeable. Many patients with hyperacidity are constipated, but the writer has not seen as yet convincing proof that any relief to the constipation is attended by an actual reduction of hydrochloric acid values, although the patients may feel subjectively relieved.

Nervous Causes.—The influence of the nervous system upon gastric digestion is generally recognized and it is well known that hyperacidity is a concomitant symptom of neurasthenic and psychasthenic states. This disorder is extremely prone to complicate mental disease, hysteria and epilepsy. von Noorden has noted its frequency in melancholia. Psychic influences frequently induce an attack in nervous individuals. Worry, undue excitement or outbursts of anger are often followed by the symptoms of the gastric disorder. There is no doubt that mental strain and worry are important etiological factors in the production of this ailment. It is not uncommonly observed during convalescence from surgical operations. Any cause which reduces nervous tone, such as poor hygiene, bad ventilation, or physical strains, may induce hyperacidity, especially if the patient be temperamentally susceptible to such influences. Individuals with broad costal angles are not, as a rule, thus susceptible, but those with acute costal angles and the other stigmas of the enteroptotic habit are especially liable to the disorder, and it is in these enteroptotic patients that the production of hyperacidity after nervous strains and worries seems most regular and certain.

The writer has no desire whatever to minimize the importance of nervous strain and of lowered nerve vitality as causes for the hyperacid state of the stomach, but he cannot believe that these neurasthenic and psychic influences actually produce an uncontrolled excitability of the secretory nerve supply of the stomach that results in the overproduction of gastric juice. It would seem more probable that these conditions of lowered nerve tone are accompanied as part and parcel of the symptom-complex by atony of the gastric wall, and that to the atony the hyperacidity is due.

Symptoms.—In discussing the symptomatology of hyperacidity it is important to separate the cases of secondary hyperacidity due to

demonstrable organic disease of the alimentary tract from those of the primary or functional form, for the reason that in the secondary group the symptoms of hyperchlorhydria are so intermixed with those of the original causative malady that the resulting symptom-complex is often exceedingly confusing. The following description of symptoms includes therefore only those of the primary form. Hyperacidity due to demonstrable organic disease is discussed under the heading of the disease to which it is secondary. Much of the confusion which results from the reading of certain medical essays on this subject might have been averted had there been made this division of the subject into these two groups.

Symptoms of Primary or Functional Acidity.—The majority of patients give no definite symptoms, as the disease runs a latent course and is discovered only by a routine examination of the stomach contents. In these cases we have no means of determining whether the process is a temporary one, present only at the time of the examination, or one of longer duration. In the patients who present symptoms of indigestion that may be attributed to the excessive acidity, only a small number give symptoms that may be referred to the stomach itself. By far the greater number complain of intestinal indigestion characterized by abdominal discomfort and distress, by irregularities in the action of the bowels, or by symptoms of intestinal toxemia, such as headache, mental depression, and the symptom-complex ordinarily described as biliousness. The various symptoms, gastric, intestinal, and toxemia, will now be described in more detail.

Gastric Symptoms.—Heart-burn is a symptom which is generally indicative of hyperacidity. It is, as its name signifies, a burning feeling referred to the epigastric, substernal, or cardiac areas, or to the lower part of the neck or throat, usually radiating upward, occasionally to the back, but never downward. Properly speaking it is not a pain, but a feeling of distress. This distinction is an important one. The burning distress comes in simple hyperacidity only during the height of digestion and should subside as the stomach empties itself. Heart-burn appearing later than three or four hours after eating suggests hypersecretion, and if complaint is made of burning distress during the latter part of the night or early morning, hypersecretion with motor error of the stomach, probably from pyloric narrowing, may be inferred. The distress is regularly relieved by eating, by drinking alkaline solutions, or emptying the stomach; but it is rare for the heart-burn to be sufficiently annoying to cause the patient to induce vomiting for its relief. If such be the case ulcer is more probable.

Heart-burn has generally been considered due to the irritation of the gastric mucosa by overacid stomach contents, but as it has been .

proved that the gastric and esophageal mucosa is absolutely insensitive to hydrochloric acid even in far greater concentration than is ever found in health or in disease, it is evident that at present its causation is obscure. Furthermore, the intensity of the heart-burn gives no correct inference whatever as to the degree of acidity that exists. Total acidities of 90 and over may not produce the least amount of discomfort, while, on the other hand, very considerable distress may be present with normal or diminished acidity, or even with achylia.

Pyrosis, or the rising of acid fluid in the mouth, is an infrequent complaint and, properly speaking, should not occur with hyperacidity that is not complicated by hypersecretion. Patients with varied gastric disorders often complain of sour food coming up into the mouth during digestion as acid as lemon juice or vinegar, and are led to regard their digestion as abnormally acid, not knowing that the gastric digestion is normally acid and that after all they are suffering only from regurgitation of food in a natural state of digestion akin to the "spilling" of babies after the taking of food. Unless reassured on this point they may do themselves harm by unnecessary medication and restriction of diet.

The raising of acid fluid unmixed with solids indicates regularly a hypersecretion, especially when it is most marked toward the latter part of the digestion when the food is leaving the stomach. Acid-rising in the fasting state does not occur with simple hyperacidity. Flatulence accompanies many of the cases and is almost always due to swallowed air. The amount of gas or wind is usually proportionate to the degree of gastric atony that is present. In the writer's experience hyperacidity without atony is not accompanied by gas, but in hyperacidity with atony, flatulency is regularly present.

A sense of fullness, uneasiness, or burning in the epigastrium which may culminate in an expulsion of wind from the stomach is common in hyperacidity. These symptoms are due to the fact that an excess of hydrochloric acid increases the peristaltic power of the stomach and at the same time induces spasm of both the pyloric and cardiac sphincters, the combined result of which is to raise the intragastric pressure and cause discomfort. This theory, however, does not explain why one individual with marked and persistent hyperacidity will not present any abdominal sensations, while severe epigastric distress and heart-burn may occur in those whose gastric analyses show a normal or even diminished acidity. It has been, therefore, supposed that the susceptibility of the stomach to free hydrochloric acid varies considerably in different individuals, and that many people are able to bear with impunity degrees of acidity that would produce severe suffering in others. Experimental proof of the correctness of this theory is, however, lacking.

Although a sudden increase of intragastric pressure may produce discomfort and distress, actual pain is exceedingly rare, and its occurrence, especially if recurring at stated intervals, should regularly suggest the strong probability of an organic cause, such as ulcer, gall-bladder disease, or chronic appendicitis. The majority of writers speak of pain, often to the point of agony, necessitating the use of narcotics, as a regular accompaniment of hyperacidity, and even the most conservative diagnosticians assert that simple hyperacidity without demonstrable organic disease in the stomach, such as ulcer or cancer, may at times produce painful sensations. Fenwick claims that in every chronic case of hyperchlorhydria the acidity gradually subsides until subacidity is attained, while at the same time painful sensations are increased rather than diminished, and that the administration of bicarbonate of soda aggravates rather than relieves the suffering. He adds that in every case of this nature in which he has seen the stomach opened for exploration, the mucous membrane was purple, swollen, and covered with superficial hemorrhages or erosions, while microscopical examination showed interstitial gastritis of the kind that is produced by chemical irritants, and he believes that this severe diffuse gastritis renders the stomach intolerant of any degree of acidity, of alkalies or even of food itself.

The author has had no experience with cases of this clinical type and doubts if such a course is observed apart from those cases in which painful acidity is the clinical evidence of organic demonstrable disease. Hyperacidity changing into subacidity or anacidity associated with epigastric pain which becomes progressively more severe and continuous and unrelieved by alkalies, has in the writer's experience turned out to be chronic gastric ulcer undergoing malignancy. The writer is extremely skeptical as to the occurrence of actual pain in primary or functional hyperacidity. In his experience patients with hyperacidity in whom organic disease can be excluded, rarely, if ever, complain of any painful sensations or even discomfort during the digesting period.

Negative Gastric Symptoms.—The negative gastric symptoms of hyperacidity are important.

1. Nausea is not a symptom of hyperacidity and is never observed in uncomplicated cases.

2. Vomiting is seldom if ever spontaneous, but it may be induced for the relief of gas and epigastric distress. There are, however, many people who are intolerant of any degree of gastric discomfort, and who have learned early in their career the trick of emptying their stomach on slight provocation. This voluntary and often totally unnecessary emesis may be quite misleading to the physician unless by a careful inquiry the emesis habit is discovered and the fact elicited

that the symptoms for which the emptying of the stomach was suggested were exceedingly slight. Such vomiting possesses very little clinical significance unless the suffering for the relief of which it is induced is sufficient to warrant it.

3. The appetite is either unchanged or increased, rarely diminished.

4. Voluntary reduction of food by the dread of subsequent pain and distress is not noticed in simple hyperacidity, but is far more significant of ulcer or of extreme degrees of atony, the former giving rise to pain which the patient desires to avoid, the latter to distress, heaviness, and gas within the stomach.

5. Hemorrhages, either visible or occult, do not occur in uncomplicated hyperacidity. When present ulcer or erosions should be suspected. Small hemorrhages with hyperacidity in those of adult years should suggest cancer.

6. The general condition remains unchanged. The strength and body nutrition are unimpaired.

Intestinal Symptoms.—The bowels may remain normal throughout the course of the ailment, although the majority of patients are more or less constipated. In chronic cases attacks of diarrhea may supervene from time to time accompanied by intestinal discomfort and flatulence and are often treated as due to chronic colitis without any consideration being paid to the primal cause for the complaint.

Intestinal indigestion may produce symptoms in many ways. Abdominal distention and discomfort are the most common symptoms present and are usually most prominent two or three hours after meals when intestinal peristalsis begins. The distention is generally diffused and symmetrical, the distress is most marked in the middle and lower abdominal zones. These intestinal symptoms may be the only evidence of hyperacidity which the patient presents, and the origin of the malady in the stomach is often overlooked. It cannot be too strongly insisted upon that in every case of so-called intestinal indigestion, an examination of the stomach contents should be made. In many patients with hyperacidity there is an interference with the intestinal digestion of a nature that we are at present unable to determine, but which is characterized by symptoms of a mild toxic nature, which for want of a better term we designate as auto-intoxication.

Headache is the principal symptom and may occur in one of two forms:

1. There may recur day after day a dull heavy ache without any characteristic localization, generally more marked in the morning and passing away as the day progresses. As a rule the patient feels heavy, dopy, and disinclined to either mental or physical exertion. The bowels are usually constipated, the tongue coated, and the breath

offensive, the complexion often taking on a sallow hue. These patients call themselves "bilious" and resort to never-ending medication.

2. There may be a dull, boring pain starting in the eyeball over one or the other side and gradually becoming hemicranial in type. The headache may be more generalized toward the close of the attack. In severe instances of this type the headache may be sudden, sharp, and generalized, and accompanied by photophobia and intolerance to noise. Nausea and vomiting may occur, but the vomiting of large quantities of acid fluid as sometimes described does not appear in these cases.

Diagnosis.—Physical Examination.—Physical examination is usually negative. There are no characteristic signs. There may be a slight tenderness in the epigastrium during the acme of the distress, but this is negligible from a diagnostic standpoint. No examination is complete that does not thoroughly investigate the motor power of the stomach and the condition of the gall-bladder and appendix. The physical signs of ulcer and cancer must be repeatedly searched for.

Gastric Analysis.—Examination of the gastric contents is absolutely necessary for the diagnosis of hyperacidity, as it is impossible to diagnose the malady by subjective symptoms alone. Examination should be made both in the fasting and the digesting state.

Fasting Stomach.—The fasting stomach should be empty of both acid fluid and food remains. Quantities of fluid under 30 c.c. are negligible but exceeding this amount indicate that some organic complication is present. Hyperacidity never passes into food stasis unless due to organic or spasmodic closure of the pylorus.

Test Breakfast.—The gastric contents aspirated one hour after the taking of the ordinary Ewald test breakfast usually show to the naked eye no departure whatever from the normal. The breadstuffs are finely chymified and homogeneous. There is usually no increase in the amount of gastric mucus. Upon settling, the contents separate into two layers, the supernatant layer not exceeding the depth of that of the layer of solids beneath. This limitation in the amount of free fluid differentiates between hyperacidity and alimentary hypersecretion—a totally different disease.

The total acidity usually ranges from 65 to 90, rarely reaching this latter point. Acidities over 90 are almost invariably associated with closure lesions, spasmodic or organic, of the pylorus, and in these cases hypersecretion is usually encountered as well. Free and combined hydrochloric acid are present, about 20 points of the total acidity being taken by the combined acid, 8 points by acid salts, the balance by free acid. Lactic and other organic acids are absent. Reactions for peptones show normal or excessive proteid digestion. There is a natural

interference with the digestion of starches, the gastric contents fail to exhibit the usual reactions for maltose while those for erythrodextrin and amidulin are well-marked. *Sarcinæ* are not present except in the cases complicated by stenosis of the pylorus and food retention.

Stool examinations in hyperacidity with intestinal or auto-intoxication symptoms seldom afford much if any clue to diagnosis. There may be small clumps of undigested starch granules, often bile-tinged, enclosed in a mucous capsule, indicating a catarrhal lesion high up in the small intestine, which is often the result of excessive gastric acidity. Fermentation of the stools according to the method recommended by Schmidt and Strassberger has to the writer been totally inadequate for diagnostic purposes. The presence of mucus in the stools, either enteric or colonic in origin, is of little diagnostic importance. With intestinal toxemia indican is usually present in increased amount in the urine, although indicanuria may be present without intestinal or toxic symptoms, while, on the other hand, these complaints may occur to a distressing degree without indicanuria beyond normal limitations. Too much stress, therefore, should not be laid upon this test.

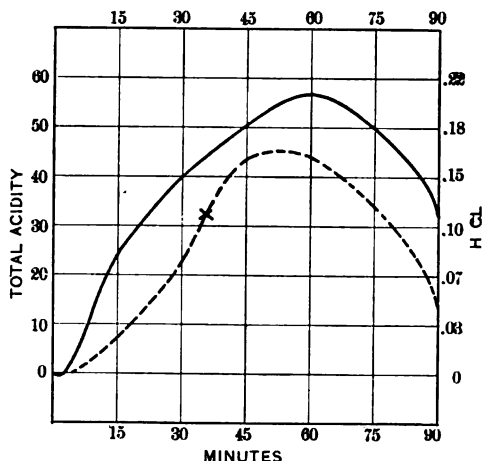
Larval Hyperacidity.—To explain the cases in which symptoms of hyperacidity are present, but in which the test breakfast shows normal or diminished acidity, Strauss suggests the following theory: The introduction of food into the stomach is followed by a pouring out of gastric juice of a definite acidity that is constant in all cases and is always stronger in concentration than is necessary for the digestion of the food. To bring this hyperacid fluid down to the desired dilution, there is poured into the stomach a neutral fluid which is called "thinning fluid." The final gastric juice is thus an admixture of the first overacid secretion and the second thinning secretion. The test breakfast at the expiration of one-half hour shows an extreme degree of hyperacidity, while examination at the expiration of an hour shows the acidity to be reduced to normal or even below the normal limits. To these cases he gives the name "Larval Hyperacidity."

The following diagram from an article by Friedenwald¹ shows clearly what is supposed to occur in these cases. This writer is a strong advocate of Strauss' theory of larval hyperacidity, and in the article just cited he gives the record of 6 cases he himself has investigated. It is quite evident, however, that Friedenwald was not dealing with simple hyperacidity but with alimentary hypersecretion, for in his cases the amount of test breakfast abstracted was excessive, ranging from 215 to 365 c.c., the bulk being composed of fluid so copious that the depth of the supernatant layer was four times that of the underlying layer of digested breadstuffs.

¹ Amer. Jour. Med. Sci., August, 1911, p. 160.

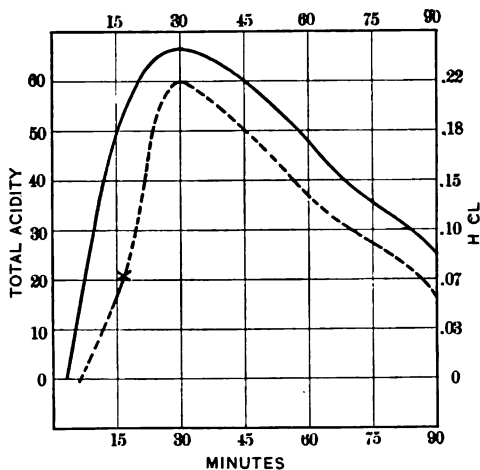
Differential Diagnosis.—No diagnosis of hyperacidity or hyperchlorhydria should ever be made unless all organic conditions are positively

FIG. 97



Curve of acidity in a normal case of hyperacidity after an Ewald test breakfast. Solid line = total acidity; dotted line = HCl; X = free hydrochloric acid. (Friedenwald.)

FIG. 98



Curve of acidity in a case of larval hyperacidity after an Ewald test breakfast. Solid line = total acidity; dotted line = HCl; X = free hydrochloric acid. (Friedenwald.)

excluded. If an exclusion of these organic causes for the ailment cannot be made, the diagnosis should be made tentatively with a view of arriving at a more definite conclusion as the case develops.

Diagnosis from Hypersecretion. — (a) Continuous hypersecretion should be suspected whenever symptoms of hyperacidity appear at a time when the stomach should normally be empty, and the diagnosis may be made with absolute certainty by the constant presence of over 30 c.c. of acid fluid giving reactions for free hydrochloric acid, in the fasting state.

(b) Alimentary hypersecretion may cause distress during the digesting period, as in simple hyperacidity; but the diagnosis can be made without difficulty by the test breakfast, which is excessive in quantity and of fluid consistency, separating into two layers on standing, the supernatant fluid being more than twice the depth of the sedimentary layer.

Diagnosis from Ulcer.—The diagnosis between simple hyperacidity and ulcer is often difficult. The problem may be simplified by the following considerations:

(a) Hyperacidity that is accompanied by pain is not functional, but is due regularly to an organic cause, which may be ulcer, cancer, gall-bladder, or appendix. The diagnosis of ulcer is then worked out by exclusion, although it often happens that a positive diagnosis can only be made by exploration.

(b) When hyperacidity is accompanied only by heart-burn or distress the differential diagnosis is more difficult. The more severe the discomfort the greater the possibility of there being an organic cause for the ailment.

(c) Hyperacidity symptoms occurring toward the close of digestion or when the stomach should be empty are not of functional origin, but are more usually due to ulcer or to appendicitis than to any other organic cause.

(d) Symptoms of hyperacidity that are aggravated by errors in diet may be considered as presumable evidence of ulcer. In ulcer the symptoms are usually relieved by a few days of milk diet, while in hyperacidity this cessation of symptoms is but rarely observed.

(e) Symptoms of hyperacidity running a prolonged course unrelieved to any great extent by treatment are in all probability due to ulcer, provided that chronic appendicitis and lesions of the gall-bladder can be excluded. Paterson¹ operated on 50 cases of persistent hyperchlorhydria, and in every instance found that there was an organic lesion either in the stomach or duodenum, gall-bladder or appendix.

(f) Occult blood in the gastric contents or in the stools is rather against functional hyperacidity, but too much reliance must not be placed upon these tests, as hyperacidity may be complicated by pore-

¹ Quoted by Moynihan, *Lancet*, January 6, 1912.

like erosions from which the bleeding takes place without any gross organic lesion being found.

Diagnosis from Cancer.—Cancer at its onset may be mistaken for hyperacidity, especially if there is carcinomatous degeneration of a chronic ulcer at or near the pylorus, or early cancer of the gastric wall with greatly impaired motility. Hyperacidity with cancer is far more frequent than is ordinarily supposed. Favoring cancer are the anorexia, advancing chloranemia and weakness in a patient of middle age who has ordinarily been able to eat with relish and without discomfort, occult hemorrhages, increasing food-stasis, and in the later stages of the disease the physical evidences of tumor and possibly of metastases.

Diagnosis from Gall-bladder Infections and Gallstones.—Gall-bladder infections and gallstones may produce symptoms of hyperacidity in one of two ways:

(a) There may be a reflex hyperesthesia of the stomach. The patient will complain of heat and burning in the substernal or epigastric region, half to one hour after eating and will say that his stomach is "scalded with too much acid." In these cases there is an intolerance for hot drinks that is quite characteristic. Soup, tea, and coffee must be cooled before they can be swallowed without producing a burning pain in the esophagus. Immediate relief follows a few sips of cold water, or the taking of soda. That these symptoms are not due to actual hyperacidity is shown by the fact that in many instances the gastric contents withdrawn at the time of the greatest distress may show normal or even subnormal acidity. It is difficult to explain these cases after the experimental work of Pawlow, Hertz, and others, which seems to prove the absence of all painful sensations in the esophageal and gastric mucosa, when bathed in even stronger solutions of hydrochloric acid than are known in health or disease. The fact remains, however, that such a distressing heart-burn and an intolerance for hot fluids clinically does exist and many continue with or without physical signs of gall-bladder diseases for months, ceasing only when a gallstone is passed or after the gall-bladder has been removed or drained from obvious outbreak of gall-bladder infection.

(b) Gall-bladder infections or gallstones may produce an actual and demonstrable hyperacidity by inducing reflex pylorospasm with delayed or impaired food exit. The gall-bladder lesions may be clinically obscure or even latent. The importance of repeated examinations of the gall-bladder cannot be too strongly emphasized in all derangements of gastric secretion whether of hyperacidity or anacidity. A carefully taken history will usually show that there have been localized attacks of discomfort over the gall-bladder. Furthermore, the symp-

toms show an irregularity in the time at which they appear after the taking of meals, which is not the rule with hypersecretion or ulcer.

Diagnosis from Diseases of the Appendix.—Diseases of the appendix, especially the chronic form of obliterative inflammation, may produce reflex pylorospasm and hyperacidity, and are usually though not invariably accompanied by epigastric pain. In some instances the pylorospasm is increased by the formation of minute erosions and is occasionally complicated by hematemesis. In these cases there is apt to be considerable tenderness over the pyloric portion of the stomach. It is important to remember that the local signs of chronic appendicitis may be trifling or even entirely absent, nor may there be in the history any evidence indicating previous attacks of inflammation. *In every case of prolonged or painful hyperacidity chronic appendicitis must be considered a possible cause, even in the absence of definite physical signs.*

Diagnosis from Hyperacid Gastritis.—The symptoms of hyperacid gastritis, as a rule, are more influenced by improper diet and abuse of alcohol than is the case with hyperacidity, although this rule is a poor one to rely upon, as hyperacidity that is due to ulcer may be subject to fluctuations depending entirely upon dietetic errors. Much valuable information is given by gastric analysis. In gastritis the fasting stomach contains usually, but not invariably, a definite quantity of gastric mucus, usually of an acid reaction, and often containing starchy food remains that may be detected by the microscope but not by the naked eye. The test breakfast is scanty, the food is intimately admixed with tenacious gastric mucus and does not readily separate into the fluid and solid layers as does the test breakfast of hyperacidity.

Lavage in gastritis brings glairy mucus, while this is not the case with hyperacidity. It should be remembered, however, that chronic gastritis may coexist with hyperchlorhydria due to other causes, and it is safe to say that a larger number of the cases of so-called chronic acid gastritis are really chronic ulcer of the stomach.

The following practical rules for diagnosis are suggested.

1. Do not make a diagnosis of hyperacidity until all organic lesions are excluded, and even then be prepared, with open and unbiased mind, to change the diagnosis to one that is more definite and distinctive should other symptoms and physical signs appear.

2. Do not make the diagnosis of hyperacidity without examinations of the fasting stomach by a tube. The presence of acid fluid or of food remains, or of any considerable amount of acid mucus should exclude the diagnosis.

3. Do not make the diagnosis of hyperacidity simply because the patient is nervous or neurotically hypersensitive.

4. Do not make the diagnosis of hyperacidity should the previous clinical history suggest attacks that may point to appendicular or gall-

bladder disease, or should the results of the physical examination be such that these lesions are probable.

5. Do not make the diagnosis of hyperacidity in cases accompanied by epigastric pain whether dependent or not upon the taking of food. Especially should this diagnosis be avoided if the pains occur at a regular period after eating.

6. Do not make the diagnosis of hyperacidity if hemorrhages from the stomach or intestines are present, either visible or occult. The examination of the stools for occult blood is a routine examination in these cases that should never be neglected.

7. Do not make the diagnosis of hyperacidity in cases accompanied by repeated vomiting, especially if the vomiting be of the abundant acid fluid indicative of hypersecretion.

8. Do not make the diagnosis of hyperacidity when the symptoms occur at a time when the stomach should be empty.

9. Do not make the diagnosis of hyperacidity in the event of the test breakfast settling into layers, the supernatant fluid being more than twice the depth of the sedimentary layer. These are the cases of alimentary hypersecretion with which hyperacidity pure and simple has nothing to do.

10. Do not make the diagnosis of hyperacidity in cases attended with anorexia, with nausea, with advancing anemia, and with progressive loss of flesh, especially if the patient be of adult years, with or without a previously good digestion.

11. Do not make the diagnosis of hyperacidity without mental reservation in those over forty-five who complain of this disorder for the first time.

12. Do not make the diagnosis of hyperacidity in any case, no matter what the symptoms may be, without corroboration by gastric analysis.

Course.—The course of the disease depends upon the nature of the exciting cause, so that we have all variations from a discomfort that is ephemeral and temporary to a harassing ailment extending continuously or intermittently for months or years. Continuous hypersecretion almost invariably is due to some organic lesion, usually ulcer or chronic appendicitis.

Prognosis.—The prognosis of hyperacidity is good for life but uncertain as to duration. Being after all only a symptom that arises from varied causes, the prognosis depends entirely upon the tractability of the underlying disease.

Treatment.—Hyperacidity is only a symptom, and its treatment, therefore, is that of the condition to which it is due. The diagnosis after all is of the first importance. Hyperacidity due to ulcer, gastric or duodenal, to lesions of the gall-bladder or appendix, may be temporarily relieved by alkalies, but the real treatment is naturally that of

the ulcer, gall-bladder or appendicular disease. It is unfortunate that so much valuable time is wasted, physical suffering prolonged and the lives of the patients kept in jeopardy by perpetual tinkering at the symptoms by lavage and alkalies when a more radical treatment is necessary. Hyperacidity accompanying atony rarely gives rise to any symptoms that call for relief, nor would it be of much use to treat such a case on purely symptomatic principles without going straight to the root of the matter and rectifying the underlying atony. If these principles be clearly understood we may now consider the means for relieving the pain or the distress which the hyperacidity causes, by purely symptomatic treatment, irrespective of the actual underlying cause.

Medical Treatment.—The chemical antidote for acidity is an alkali. Bicarbonate of soda is perhaps the most useful, certainly the one most commonly employed for this purpose. Soda may be given in doses from one-fourth to one-half a teaspoonful in water at the time of the greatest distress, or soda mints or the lozenges containing 15 to 20 grains of the preparation may be carried in the pocket and slowly dissolved as needed. There is much popular objection to the use of the remedy, but the writer believes that the lesser evil lies in the reduction of excessive acid and sees no objection to its reasonable use even over long periods of time. Alkaline earths and carbonates are of service. Magnesium oxide or the calcined magnesia is a valuable antacid and serves, moreover, to produce a laxative effect. Subcarbonate of bismuth is most serviceable in irritative conditions of the stomach such as accompany ulcer, or when the hyperacidity is accompanied by vomiting or diarrhea, and may be combined in varied proportions with magnesia and soda. Sodium citrate in teaspoonful doses may be given between meals, often with brilliant results. Calcium carbonate may be used either in powder form or in suspension.

The following prescriptions have been of service, and the ingredients can be arranged in a variety of ways to meet individual requirements.

R—Magnesia usta gr. iiij
 Bismuth subcarbonate gr. v
 Sodii bicarbonate gr. xv

M. Sig.—Such a powder two hours after eating.

R—Cerium oxalate,
 Bismuth subcarbonate āā gr. v
 Magnesia usta gr. iiij
 Sodium bicarbonate ad gr. xxx

M. Sig.—Such a powder whenever heart-burn occurs.

R—Orthoform,
 Bismuth subcarbonate āā gr. v
 Mist. cretæ comp. ad ʒj

M. Sig.—Teaspoonful in a little water for indigestion.

It must be remembered, however, that these alkaline powders are merely symptomatic in their use.

Alkaline waters may be recommended, although if atony, gastrop-tosis, or pyloric contraction be present, they should be allowed only in minimum doses *if at all*.

The best waters for such purposes are the Saratoga Vichy, Witter Water Spring, or in Europe Fachingen, Giesshübel, or Vichy. These waters are to be preferably warmed. They may be taken before break-fast, or at any time that an antacid is needed for relief. The writer has seen no beneficial effects from their use as a "cure," but has noticed considerable betterment in the distress of hyperacid patients when the water is taken simply as a temporary means of relief, but they possess no advantage over the alkaline powders ordinarily prescribed for the same purpose.

Carlsbad water may be of service in hyperacidity due to ulcer or to gall-bladder infections, or resulting from chronic gastritis. In these instances a glass of Sprudel as hot as can be sipped should be taken on arising in the morning, at least three-quarters of an hour before breakfast, and possibly half such a dose may be taken an hour before the evening meal. The quantity taken should be so limited that only one loose movement of the bowels results.

Either the imported water may be taken, or the artificial salt may be used suitably diluted. The powder obtained by the evaporation of the Sprudel water at the spring is the best form in the market, and is imported by Eisner and Mendelson in this country. The dose is a level teaspoonful to a tumbler of water. Sodium chloride waters, such as Hawthorne and Congress water, and in Europe Kissingen, Homburg, Soden, and Wiesbaden are positively contraindicated.

Belladonna and atropine have been recommended because of their effect in inhibiting excessive secretion, but unfortunately to produce such a result the drug must be given in such doses that unpleasant physiological effects may require its discontinuance. The writer has employed both small repeated doses of the tincture of belladonna (Mij to v, t. i. d.) or minute doses of atropine (gr. $\frac{1}{40}$, t. i. d., or gr. $\frac{1}{80}$ every three hours), but has generally been disappointed in the results. The remedy has usually been worse than the disease.

Olive oil has been recommended, owing to its supposed inhibitory effects upon gastric secretion. A tablespoonful may be administered half-hour before the meals or half a wineglass (3j to 3ij) may be given on retiring. When hyperacidity is due to pylorospasm from gastric or duodenal ulcers or abrasions the results of the oil treatment are generally very good, but in hyperacidity otherwise induced, the treatment has seemed most disappointing.

Silver nitrate has been long regarded as serviceable in reducing acidity. Whether or not actual reduction in total acidity of subsequent test breakfasts can be demonstrated is problematical, but the drug certainly does seem to relieve in great measure the discomfort of the disorder. Silver nitrate may be given in solution, capsule, or by lavage. The dose of the drug by mouth is about gr. $\frac{1}{2}$, t. i. d., and it may be given either in solution or in capsule. A useful method is that by cycles of gradual increase in the dose. For three days gr. $\frac{3}{8}$ are given in distilled water half-hour before the meals—for the following three days gr. $\frac{4}{8}$ are similarly given, while in the last three days of the cycle the dose is increased to gr. $\frac{5}{8}$. After the lapse of three days the cycle is to be repeated. Should diarrhea occur the dose should be reduced or the treatment abandoned. The following prescription may be employed:

R—Argenti nitrat. gr. xxxij
 Aq. destillatæ ℥ij

M. Sig.—5 minims contain gr. $\frac{1}{4}$ of silver nitrate. Dose 15 to 25 drops well diluted with distilled water one-half hour before meals.

Lavage with $\frac{1}{3000}$ solution of the nitrate may be found distinctly serviceable. The stomach should first be washed with ordinary water until the return flow is clear, and then with about 2 pints of the silver solution, taking care that as little as possible of the solution remains in the stomach. This treatment may be given every second day, and should be discontinued temporarily if diarrhea occurs. If seemingly beneficial the strength may be gradually increased to 1 to 1500, but greater concentrations than this are not to be used. In making a solution of 1 to 3000, ℥j of the above prescription to the pint of water is sufficiently accurate.

Aluminum salicylate has been highly recommended by Rosenheim of Berlin in doses of half to one teaspoonful shaken with water about half to one hour before meals. The drug is in the market under the name of Neutralon (Kaulbaum)—a fine odorless and tasteless powder, insoluble in water. The writer has had no experience with this drug.

Of late the use of hydrogen peroxide has been warmly indorsed. Fifty cubic centimeters of a 3 per cent. solution are made up to 300 c.c. with water, making approximately 0.5 per cent. solution. A similar dilution may be obtained by using ℥j of the 3 per cent. peroxide in a tumblerful of water. A glassful should be taken an hour after meals. Goodman, writing from the laboratory of the late Dr. John H. Musser, is a strong advocate of this treatment. In the author's cases considerable relief to the heart-burn and distress may be expected, although he has not been able to satisfy himself that any permanent reduction

in the total acidity of the gastric contents follows the treatment. The objection to the water is the fishy taste. Similar results may be obtained by perhydrol in teaspoonful doses, although less certain than with the peroxide water.

Hyperacidity due to atony even after it has resisted ordinary treatment may be relieved by large doses of the tincture of *nux vomica*. The drug must be given in ascending doses and in large quantities, and generally over long periods of time, usually four or five months. The late Dr. John H. Musser recommended that in most cases 60 or more drops three times daily should be given, although the point of tolerance should be computed for each individual. *Nux vomica* and not strychnine should be used.

Dietetic Treatment.—The choice of diet depends upon the underlying cause for the hyperacidity. If ulcer be the primal cause, the diet is that of ulcer, while in atony and atonic gastroptosis the diet appropriate to these conditions is to be enforced. When chronic appendicitis is the cause, no lasting benefit can result from diet regulations, although attacks of pylorospasm may in all probability be rendered less frequent and less severe by the enforcement of a diet that is mechanically bland and unirritating. In general starches should be somewhat reduced and the fats, in the form of cream and fresh butter correspondingly increased. Such a diet may be constructed on the following scheme:

BREAKFAST:

None: Coffee not advised; no tea allowed; no coarse cereal, such as oatmeal or cracked wheat; no bread crusts, dry toast, or hot bread; no salt fish or potatoes.

Allowed: Cocoa, with cream and sugar. Fine cereal, such as cream of wheat, farina, etc. Soft parts of bread, milk, or cream toast. Crackers thoroughly masticated. Butter, preferably unsalted, to be taken as freely as possible. Creamed or minced chicken; fresh fish; soft-boiled or poached eggs.

LUNCHEON:

Purée or cream soup of any kind, made without meat stock; no other soups allowed. Lamb; simply prepared ragout; lean broiled or boiled ham; fish, chicken, oysters in any form. Fowl, except domestic duck or goose. Mashed or baked potatoes; spaghetti or macaroni. Any vegetable that can be put through a purée sieve allowed. Any green vegetable (such as string beans) may be taken if tender, not if tough. Salad, with French dressing, made with lemon. Farinaceous desserts, such as rice pudding, corn-starch, blanc-mange, custard, etc. No ice-cream or ices. No fruit of any kind. Alcohol not allowed in any form.

Cheese: Camembert, Roquefort, Cream, Brie, Neufchatel, pot-cheese.

DINNER:

Same variety as for lunch.

Between meals may be taken: Choice of custard, junket, raw eggs or egg and milk shake, chicken or meat sandwich; malted milk, cocoa. Milk in the glass not allowed.

Seasoning, such as pepper, salt, paprika, etc., should be reduced to the minimum.

Water should be cool but not iced; Celestins or Saratoga Vichy, Fachingen, Apollinaris or Giesshübler preferable to plain water; when these waters cannot be obtained, may drink water containing one quarter of a teaspoonful of bicarbonate of soda to the glass.

The danger is that the dieting is often overdone and the patients are so restricted that they are not allowed enough food to keep up their general strength. In the writer's experience this danger is real, not fanciful, and especially in the atonic cases much harm may ultimately result.

The writer has seen no special results from the adoption of a salt-free diet.

CHAPTER XVIII

ACHYLIA

UNDER the term "achylia gastrica" are included those conditions in which hydrochloric acid is absent both in a free and combined form from the gastric juice. Properly speaking the term should be limited to those cases only in which neither acid nor ferments are found in the stomach contents, or as Martius expresses it, "When the gastric juice is deficient in all of its ingredients." Clinically, however, we find very few cases in which this double deficiency exists. Acid secretion and ferment secretion are two distinct results of glandular activity and quite independent of each other. In general it may be said that the acid secretion is far more easily checked than is the elaboration of the ferments, so that we usually find that even with entire absence of hydrochloric acid the ferments are present, although they may be secreted in diminished amounts. It is only in the most extreme instances of atrophy of the entire glandular elements of the stomach that total absence of ferments exist, so that if the term were strictly used, it would apply to only a very small proportion of the cases in which tests show an absence of acid secretion. The term, therefore, is used clinically in the broader sense of implying only an absolute hydrochloric deficiency in the gastric juice, and is very convenient to use as it covers a class of cases in which achlorhydria occurs from such a variety of causes, some definite, others obscure, that an orderly classification on pathological grounds cannot be made.

Forms.—Achylia may occur both in malignant and in non-malignant disease.

Malignant Achylia.—In 2500 private patients with stomach and intestinal disorders, 149, or 6 per cent., showed achylia that was not due to cancerous disease of the stomach. During this same period of time there were 40 cases of cancer, of which gastric analysis was made in 18. Of these 18 cases, in only 4 was there a true achylia present without evidences of malignancy in the test breakfast, such as lactic acid, blood, Oppler-Boas bacilli, or food stagnation. Therefore, of 153 consecutive cases of apparently simple achylia only 4 were malignant. These figures are interesting as tending to disapprove of the old idea that absence of hydrochloric acid meant malignancy. It cannot be affirmed too positively that the presence or absence of hydrochloric acid has very little bearing upon the diagnosis of gastric cancer. Achylia

occurring in malignant disease of the stomach is described in chapter on Cancer.

Nature and Pathology.—Achyilia may occur from a variety of causes which may be roughly grouped as follows:

1. Achyilia from inflammatory processes in the stomach with or without atrophic changes.

2. Achyilia from atrophic changes in the gastric mucosa not definitely consequent upon an accompanying inflammation.

3. Achyilia as the result of the lack of functional activity of the secretory glands of the stomach, either as a pure neurosis or reflex inhibition of function from primary forms of nerve irritation.

Achyilia as the Result of Gastric Catarrh with or without Trophic Changes.

—There is no doubt that gastritis exists in a large number of cases of anacidity. The German writers consider that this is by far the most frequent form, and that anacidity, furthermore, in gastric catarrh represents the terminal stage of the inflammation attended by the destruction of the gastric tubules, so that secretion of these products is no longer possible. This opinion is well expressed by Elsner as follows:

“In the great majority of cases achyilia is occasioned by an organic disease of the glands of the stomach due to a chronic inflammatory process of a catarrhal nature which has resulted in complete destruction of the secreting apparatus and in atrophy of the mucous membrane. It is the clinical expression for ‘anadenia gastrica.’ All injuries which can provoke chronic catarrh are equally etiological factors in achyilia. Among these may be mentioned an unsuitable and improper diet, abuse of tobacco and alcohol, defective teeth, and imperfect mastication as well as habits of hasty and rapid eating.” According to this writer the disease is more common among the poorer classes than among those in comfortable circumstances.

With these views the writer can agree in but a part only. There is no doubt that in the majority of cases of *alcoholic* gastritis the general trend of the disease is toward a progressive reduction of secretory and peptic power which becomes most marked whenever cirrhosis intervenes, and which is due to the effect of the progressive inflammation. Even in these cases, however, the clinical evidence of achyilia may not be associated with pathological changes in the gastric mucosa to account for the absence of secretory power. The glands seem normal enough to have done their work, but why they have not done so we cannot say.

In the forms of gastritis that are not due to overindulgence in alcohol, it is in rare instances only that we can obtain a clinical history of gastric catarrh. The patients are usually well nourished, and are neither enfeebled nor anemic. They deny having had previous gastric distress, nor is it possible to elicit the history of previous symptoms of heart-burn

so characteristic of the hyperacid cases. Of a certain number, gastric analyses show the presence of mucus in such quantities that the origin of achylia in a gastric catarrh cannot be disputed, but these form only a small proportion of the achylia cases, as "wet achylia" or the form of test breakfast in which undigested bread fragments are obtained floating in mucus, occurs in but 30 per cent. of the total number of cases of achlorhydria. The more usual forms of gastric contents in which only small quantities of mucus are present enveloping and infiltrating the food fragments cannot be considered as a proof of the catarrhal origin of the disease, for the reason that the secretion of mucus may be only the result entirely of the irritation of the gastric mucous membrane by food that is totally undigested by reason of a primary achylia. On the other hand, the absence of mucus cannot exclude gastric catarrh because the mucus secreting glands may be involved in the same inflammatory process that has destroyed the glands whose function it is also to secrete acid and digestive ferments.

Again it must be remembered that latency in gastric catarrh occurs even with definite and well-marked pathological changes in the mucosa, and that gastritis even if severe from an anatomical standpoint may exist for years without the least clinical evidence of its presence.

The above are the clinical reasons why it is difficult to accept without reserve the view that achylia owes its origin to severe and continued gastric inflammation.

Lange and Faber,¹ on the other hand, in a recent article, after a most careful pathological study of sections of the stomach in achylia in which postmortem change had been prevented by the intra-abdominal injection of 10 per cent. formalin solution immediately after death, conclude that while a purely functional origin of achylia cannot be disputed, it occurs but rarely—as in their experience chronic inflammatory changes in the mucosa were almost regularly present and consisted in a chronic interstitial inflammation, together with the parenchymatous changes common to gastric catarrh. Atrophic changes in their cases were not, however, prominent. In twelve cases, atrophic changes were considerable in but two, moderate in one, *insignificant* in nine. They conclude that in practically all of the cases of achylia, gastritis is present, but that the degree of anatomical change cannot serve as an indication of the amount of functional disturbance resulting from it. They allude to the fact that in acute catarrh of the stomach in which pathological changes in the mucosa are much less marked than in achylia, functional derangements may be profound. This seems to the writer to be the weak point in all the conclusions drawn from the pathological study

¹ Zeitsch. f. klin. Med., 1908, lxvi, pp. 53 and 247.

of achylia—that the degree of atrophy is not enough to explain the loss of function on any pathological basis. Moreover, it is possible that the gastric catarrh results from the irritation of undigested food within the stomach instead of being the primary cause for the achylia.

In their studies of 7 cases of achylia with pernicious anemia, Lange and Faber report that the prominent feature was inflammation of the mucosa, especially of the interstitial type, as shown by cellular infiltration throughout the mucosa, but especially well-marked near the surface, while the glandular structures were well preserved. They conclude that the prominent gastric lesion in pernicious anemia is gastritis, and that atrophy is in no sense essential.

The writer would suggest that it is not improbable for achylia to develop in various forms of toxemia, the lesions of chronic gastric catarrh being the pathological result of the toxins, while functional activity is checked by the toxic products present in the blood acting locally upon the secretory apparatus of the mucous membrane of the stomach. It is possible that the unknown toxins of pernicious anemia, together with the products of hemolysis common to this disease, may serve as an excitor of gastric catarrh and as a depressor of gastric function. The achylia that often accompanies bothriocephalus anemia may be caused in the same manner. The toxemias of chronic Bright's disease and of chronic pulmonary tuberculosis might likewise possibly cause the achylia that so often accompanies these diseases. The close connection that clinically exists between achylia and infections of the gall-bladder might according to this reasoning be explained by the toxins of the former infection acting upon the mucous membrane of the stomach.

The weak point in the writer's suggestion is the fact that after the cure of the primary disease and complete elimination of the toxins, the gastric functions are but rarely resumed. Thus, some of the cases of bothriocephalus anemia are followed by persistent achylia even after the expulsion of the parasite and the cure of the anemia.

Faber and Lange believe that whenever an achylia that has existed for years is succeeded by a return to normal secretion, there has been effected a cure of the chronic gastritis to which the achylia owed its origin. When we consider, however, that in three-quarters of the cases studied by these authorities atrophic changes were insignificant, and the amount of anatomical changes in the mucosa quite inconsiderable, we must confess that we know very little indeed, if in fact we know anything at all, about the pathology of achylia.

It is a well-recognized fact that the mucous membrane of the stomach in achylia is exceedingly vulnerable and that small bits and fragments are easily detached by the eye of the stomach tube and are found either

in the test breakfast or in the lavage water. Examinations of these fragments have frequently been made, but while a certain number show the presence of inflammatory changes, many again are apparently normal and it is significant that Lubarsch who has certainly done as much work in this direction as any other man, working in conjunction with Martius, should warn us that we are not justified in drawing conclusions about the condition of the stomach as a whole by any examination made of a small bit of mucous membrane whose previous location in the stomach is unknown.

Achylia Resulting from Atrophic Changes in Stomach.—*Achylia may result from atrophic changes in the stomach that are not definitely consequent upon inflammatory processes.* Of this form two distinct types are recognized:

1. Atrophy of gastric tubules may occur with pernicious anemia, and is generally considered at the present time to be the result of the blood changes of that disease rather than the cause for the anemia.

At variance with this view, however, are the results of pathological studies in achylia accompanying pernicious anemia by Faber and Lange who found in 7 cases sectioned by them that the prominent pathological change in the mucosa was one of interstitial inflammation and that the glands were well preserved, so that atrophy could not be regarded as an essential lesion. Further studies on this point are necessary.

It is not known whether the achylia that accompanies various forms of toxemia, such as bothriocephalus anemia, chronic pulmonary tuberculosis and chronic nephritis are to be explained by atrophy, by chronic gastritis secondary to these diseases, or by a functional inhibition of secretion.

Atrophic changes occur with cancer, either occurring locally in the stomach or in some organ, however far distant. In both these instances the cause for the atrophy is probably some form of toxemia or hemolysis at present unknown to us.

2. A very different type is the form of chronic productive inflammation and atrophy that occur in elderly subjects and is analogous in its essential nature to other forms of senile degeneration and arteriosclerosis common to those of advancing years.

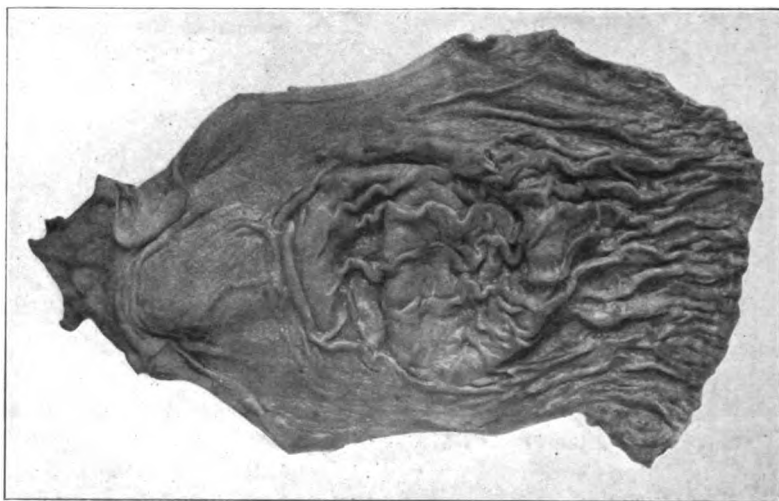
Soltau Fenwick¹ has described these cases and gives the following description of the pathological changes that are manifest. The lesions usually begin after fifty, although they may be present even before this time. The pyloric portion of the stomach appears attenuated, the rugæ absent, and the mucous membrane smooth and generally adherent to the muscular coat. There may be glistening streaks running

¹ Lancet, November 6, 1909, p. 1347.

parallel with the lesser curvature or irregular particles of this scar-like tissue over the mucous membrane near the pylorus. Extensive atheromatous changes of the aorta, coronary, and mesenteric arteries are usually observed.

Microscopical examination shows a growth of connective tissue surrounding the glands so that the latter appear unduly separated from one another. As the disease progresses, the increasing interstitial tissue twists, distorts, and compresses the glandular structures until they finally disappear, leaving the mucous membrane converted into a thin layer of fibrous tissue. The submucosa suffers from the same form of cirrhosis with obliterating endarteritis of the nutrient vessels. There are no evidences of hyperemia for any part of the process; similar lesions may be noticed in the small intestine.

FIG. 99



Atrophic form of stomach common in elderly subjects. The gross characteristics as described by Fenwick are well represented.

Fenwick estimates that 21 per cent. of those over sixty-five suffer from chronic indigestion, of 100 such cases 66 per cent. are secondary to organic disease, including 10 per cent. whose indigestion is due to hypersecretion the result of chronic ulcer, gallstones or chronic appendicitis. The symptoms of the remaining 34 per cent. are due to the progressive atrophy of the gastric mucosa just described.

Achylia was found by Stockton in 37 per cent. of his examinations in those over fifty, by Seidelin in 40 per cent. under similar conditions.

The author annexes two tables showing the relation of achylia to the various stages of life, taken from the records of his private cases.

In the first table are given the percentage in which achylia is found in the different decades among those applying for treatment for various digestive disorders.

In the writers's series of achylia

1 per cent.	occurred between the ages of 10 and 20 years
5 per cent.	occurred between the ages of 20 and 30 years
25 per cent.	occurred between the ages of 30 and 40 years
33 per cent.	occurred between the ages of 40 and 50 years
20 per cent.	occurred between the ages of 50 and 60 years
14 per cent.	occurred between the ages of 60 and 70 years
2 per cent.	occurred between the ages of 70 and 80 years

This table brings out the relative frequency of achylia in the various ages that apply for medical treatment. The number of patients over fifty or sixty who apply for treatment is relatively smaller than those of less advanced years of life because there are fewer of them.

In the succeeding table the writer has taken 100 patients in each decade who have gastro-intestinal symptoms and in whom gastric analysis has been made to see how many of these have achylia, with the following result:

Between 20 and 30 years	4 per cent.
Between 30 and 40 years	12 per cent.
Between 40 and 50 years	11 per cent.
Between 50 and 60 years	24 per cent.
Between 60 and 70 years	36 per cent.

The writer is inclined, therefore, to believe that estimates of Fenwick and Stockton are rather under the mark and that 60 per cent. of these over fifty show by gastric analyses that achylia is present, although in a large proportion of these the disorder may run its course without symptoms.

Achylia Due to Functional Derangement.—*Achylia is frequently found in cases in which structural changes in the mucous membrane of the stomach are suggested neither by the clinical history nor by the physical examination of the patient, and we are compelled to include these unexplained cases under the general heading of secretory neuroses.* There is no doubt but that achylia may exist as a purely functional derangement, and the absence of hydrochloric acid in the gastric juice may be followed, within a short period of time, by a recurrence of its secretion, without treatment, nor any change in the diet or mode of life of the patient. These cases are frequently termed "heteroachylia." The disappearance of the hydrochloric acid in these patients is usually but temporary, the patients are usually neurotic in temperament, and the recurring acidity is often abnormally excessive.

Temporary achylia may occur as the result of sudden nervous shocks, or of acute depressed mental conditions. This mode of occurrence is frequently seen in those who suffer from great nervous excitement from whatever cause, and in this condition, sit down to their meals—suffering for some hours afterward from epigastric distress, nausea, and the vomiting of the food taken at the previous meal, totally undigested and without trace of hydrochloric acid either free or combined. Temporary achylia occurs so frequently just before or during the first day of menstruation that gastric analysis should never be made at this time if it can be avoided, as the chemical findings are apt to be misleading.

Far different, however, are the cases in which achylia without known cause continues steadily and without change for months and years, for it is an established fact that persistent achylia can occur in this way without structural changes in the secreting apparatus of the stomach.

The writer remembers especially one patient in whom achylia of the dry variety had persisted to his knowledge for years. The chief complaint was of pain, due to adhesions between the gall-bladder and lesser curvature, for the relief of which an operation was performed, the patient dying on the seventh day from hemorrhage into the pons. Specimens snipped from the stomach wall at the time of operation and sections made in various parts of the organ after death, preserved from postmortem changes by the filling of the stomach with formalin solution a few moments after death, failed to reveal the least evidence of disease, and were regarded by the late Dr. Hodenpyl as absolutely normal in every particular.

The origin of these cases cannot be explained—the absence of organic changes in the stomach would seem to imply that they were purely functional, but on the other hand it is inexplicable that a person who is as apparently well and healthy as are most of the achylia patients, and without any evidences of nervous instability, should go along year after year with a nervous suppression of gastric juice as the one and only functional derangement that we can detect. It is unlike a systemic neurosis to focus itself upon one body function for so long a time, without causing other and varying functional derangements. No other secretion of the body is affected in like manner. Patients with achylia as a class are not more nervous than are a similar number of patients taken at random, neither does it seem that the predisposition of women to the disease is sufficiently great to suggest a neurotic origin. Under fifty the disease occurs in women in proportion 2 to 1, while after this age the sexes are nearly equally represented.

The view that achylia is the manifestation of a primary neurosis is supported therefore by insufficient evidence.

It has been suggested that instead of achylia being a primary neurosis, the secretion of gastric juice may be inhibited by reflex action from a primary source for irritation, at some point more or less distant, a point of view suggested by the frequent association of pathological changes in the various organs of the body generally, with the clinical evidences of achylia.

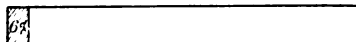
Relation of Achylia to Other Diseases.—In the following table the writer has tabulated the various pathological processes that occurred in 100 cases of achylia in his private practice:

ACHYLIA IN RELATION TO OTHER DISEASES. 100 CASES.	
Negative findings	34 cases
Gastroptosis alone	14 cases
Gall-bladder alone	16 cases
Gastroptosis and gall-bladder	4 cases
Arteriosclerosis alone	6 cases
Arteriosclerosis and gall-bladder	2 cases
Cardiac (all compensated)	3 cases
Gastroptosis and cardiac	2 cases
Fatty liver	1 case
Hypertrophic cirrhosis	2 cases
Tuberculous peritonitis	3 cases
Pulmonary tuberculosis	2 cases
Chronic appendicitis	5 cases
Stone in ureter	1 case
Dilated esophagus with cardiospasm	1 case
General paresis	1 case
Athyroidism	2 cases
Postoperative for cancer of uterus (after six years)	1 case
100 cases	

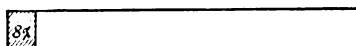
It will be seen that the associated diseased conditions cover a very large range, and are so scattered that with two exceptions no one of them is observed with sufficient frequency with achylia to have any apparent connection with this disease. For example, achylia occurs in 6 per cent. of all patients applying for treatment and in but 8 per cent. of those who suffer from chronic appendicitis. Reference to the following graphic chart will show that there is no apparent connection between the two conditions.

FIG. 100

Percentage of achylia in all patients, 6 per cent.



Percentage of achylia in appendicitis, 8 per cent.



Association of Achylia and Gall-bladder Disease.—Of more importance is the association of achylia with cholelithiasis and diseases of the gall-bladder. Twenty-two per cent. of the writer's achylia were thus complicated.

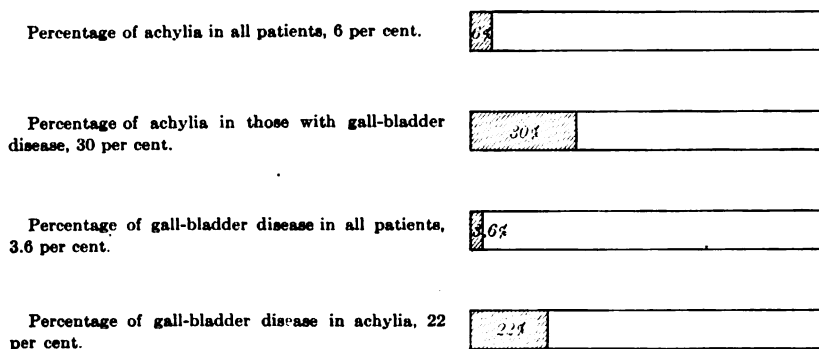
In 100 cases of gall-bladder and gallstone disease in the writer's practice achylia was found in 30 per cent. as shown in the following table:

ACIDITIES IN 100 CASES OF GALL-BLADDER DISEASE

Hyperacidity	30 per cent.
Normal acidity	30 per cent.
Subacidity	10 per cent.
Achylia	30 per cent.

The probability of there being a close causal relation between the two diseases is shown by the study of the accompanying diagrams:

FIG. 101



In the first table it will be seen that achylia complicates 6 per cent. of all gastro-intestinal patients, while with gall-bladder disease 30 per cent. of patients are thus affected. *Achylia is five times more frequent in those with gall-bladder disease than in those who are free from this complaint.*

The second table shows that the patients who apply to the specialist for gastro-intestinal disorders, 3.6 per cent. give evidence of gall-bladder infection in one or the other of its forms, while in 100 patients with achylia, 22 per cent. suffer also from gall-bladder disease. *Infections of the gall-bladder are thus six times as frequent in achylia as in those whose gastric secretions are normal.* An example of a not unusual clinical history may be given pointing to such a connection between the two diseases:

Mr. W. G. A., aged forty-seven years, is of temperate habits and careful in the choice of his food, which he masticates thoroughly.

Is not a nervous man by nature, and his life is well regulated and orderly. He has never suffered from any abdominal distress until his present illness.

Three years ago he began to suffer from recurrent attacks of gallstone colic, during several of which he became distinctly jaundiced.

Physical Examination.—The gall-bladder is palpable and distinctly tender. There is well-marked rigidity both of the right costal arch, and of the upper part of the right rectus muscle. Otherwise his examination is negative.

Fasting stomach, empty.

Test breakfast, 50 c.c., poorly chymified, of dryish consistency, with scanty quantities of mucus enveloping the bread fragments. Total acidity 12. Free hydrochloric acid absent.

In many cases cholecystitis is directly and immediately followed by the disappearance of hydrochloric acid from the gastric juice.

The following example of this may be given:

A robust athletic young man consulted the writer for a temporary indigestion, and at this time the test breakfast was normal. Two weeks later he was attacked by hepatic colic, the stone lodging in his common duct. Three days after this gallstone attack his test breakfast showed achylia. The stone was subsequently removed surgically, but although his recovery was perfect, and he is the picture of athletic manhood, eating and apparently digesting everything with impunity, at no time, now seven years since the attack, has the gastric juice ever shown the presence of hydrochloric acid in either free or combined form.

The following case is one of achylia following typhoid infection of the gall-bladder:

E. C., aged thirty-five years, was under the writer's care for many years suffering from gastropotosis and a mild degree of atony. Examination of stomach contents was repeatedly made during this time and invariably showed normal or slightly excessive acidity. In July, 1909, the patient went through a mild typhoid, complicated by pain and tenderness referred to the gall-bladder region. There was slight rigidity of the upper right rectus muscle. These symptoms continued off and on for three months after recovery from the fever, and during this time and since then, a period of three years, an absolute achylia has persisted. This sequence of typhoid fever, infection of the gall-bladder, and achylia has repeatedly been observed by the writer.

It would also seem that achylia of those past middle life might be explained by the increasing number of cases of cholelithiasis and cholecystitis during these years as well as by the occurrence of the

pathological changes of atrophy previously described—and the writer has compiled from his cases the following table:

AGE RELATIONS OF ACHYLIA AND CHOLELITHIASIS.

Of patients with achylia between the ages of 40 and 50 years, gall-bladder disease was present in 27 per cent.

Of patients with achylia between the ages of 50 and 60 years, gall-bladder disease was present in 20 per cent.

Of patients with achylia between the ages of 60 and 70 years, gall-bladder disease was present in 47 per cent.

The writer believes therefore that there is a close causal relation between gallstones and inflammation of the gall-bladder on the one hand and achylia on the other. These former conditions often run a course attended by such insignificant local symptoms and physical signs that their presence is unsuspected. Were we able to ascertain correctly the condition of the gall-bladder in all of our cases of achylia it is quite probable that many cases of obscure origin might be explained in this way. The longer we watch our individual cases of achylia the more often do we find attacks of gall-bladder infection appearing from time to time in the clinical history. Of interest in this connection is the following history, showing latency of the gall-bladder lesion until long after achylia had been recognized.

Mrs. H. T., aged fifty-four years.

The appendix had been removed eight years ago, after the third attack of acute inflammation.

Three years ago complained of indefinite pains in the upper abdomen and vague symptoms of dyspepsia. These symptoms soon disappeared, returned for a few days a month or so later, since which time she has been free from all distress. For two years her complaint has been of diarrhea in attacks lasting for several weeks at a time quite severely, having during these periods four or five loose movements limited to the morning hours. At other times she will have but one semiformal movement daily. At no time has there been nausea, vomiting, or any epigastric distress whatever.

Physical Examination.—Negative, no tenderness over gall-bladder region. Gall-bladder not palpable. No dorsal point of tenderness.

Fasting stomach empty.

Test breakfast, 50 c.c. of poorly digested bread fragments, admixed with a considerable amount of mucus. Total acidity 10; free hydrochloric acid absent.

Lab-zymogen active in dilutions of 1 to 10; inactive in dilutions of 1 to 20; lavage in the fasting state brought no mucus. Stool examination showed a mild enteritis.

The case was considered one of anacid catarrhal gastritis and was so treated.

Four months later acute empyema of the gall-bladder appeared, with purulent infection of the right pleural cavity, from which she died. It is probable that old standing cholecystitis with gallstones had been present, causing the symptoms of which she complained three years before her death and which ran a latent course and without physical signs during the time in which she was under observation, until suddenly symptoms of severe gall-bladder infection appeared and caused her death.

It is interesting to note that improvement or cure of the gall-bladder disease is not, as a rule, followed by a return of the gastric secretion.

It is possible that achylia may result from derangements of the secretion of the internal glandular structures of the body. Of 176 cases of achylia there were three patients in whom symptoms of hypothyroidism were present. In all of these rapid improvement in general condition followed thyroid feeding, although in none was this improvement followed by the return of gastric juice. It is to be hoped with increasing knowledge of internal secretions, light may be thrown upon a possible relationship of these derangements with achylia.

Association of Achylia and Gastropotosis.—It is apparently significant that gastropotosis should accompany 20 per cent. of achylia, either alone or associated with cardiac or gall-bladder disease. Many authorities in their description of gastropotosis consider that achylia in that condition is exceedingly frequent.

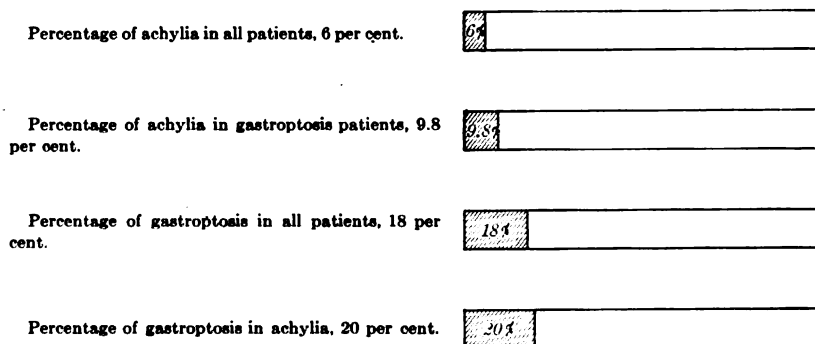
Steel and Francine consider that in gastropotosis, absence or diminution of the free hydrochloric acid is the rule—Brown, in Osler's *Practice of Medicine* states that hydrochloric acid was absent in most of the patients with gastropotosis accompanied by high grades of dilatation. The writer cannot believe that these statements are correct, nor can he see that there is any connection whatever between achylia and gastropotosis, but considers that their association is entirely accidental and is due to the fact that both diseases occur with such frequency that in a certain number of patients they are unfortunately liable to occur at the same time.

Gastropotosis occurs in 18 per cent. of all patients with gastro-intestinal symptoms and in 20 per cent. of those with achylia—a difference in frequency too small to be of importance.

Achylia occurs in 6 per cent. of all patients with gastro-intestinal symptoms, and in but between 9 and 10 per cent. of those with gastropotosis, again a difference in frequency that may be considered negligible.

These percentages may be graphically shown by the accompanying diagrams:

FIG. 102



Symptoms.—Latent Course.—In the majority of cases of achylia, gastric symptoms are absent, or if present are due to associated conditions which may coexist in the same patient, quite independently of the achylia itself, and no suspicion of its presence is entertained until chemical examination of the gastric contents is made.

The patients do not, as a rule, look or act differently from those whose digestion is normal. They are not anemic nor neurasthenic, neither are they undernourished. They live wisely, eat all kinds of food with relish and without the least distress, nor do they give in their clinical history any complaint of having any indigestion whatever. They simply have no gastric juice, and they go for months or years without its return, apparently none the worse for its absence. There is nothing in their history, mode of life, or physical examination to throw any light upon the nature of the process. We find by accident that they have achylia—more than this we do not know.

Such a clinical history may not occasion surprise if elicited from a patient in whom we suspect a functional origin for the achylia, but it is more remarkable when well-marked structural change in the mucous membrane of the stomach is apparently the cause for the complaint. In these cases of apparent organic origin the course throughout may be latent, or there may be the history of previous severe indigestion that has gradually lessened so that symptoms have become finally quiescent and the patient presumably free from all gastric error. The following case may be cited as an example of achylia without symptoms, apparently due to structural changes following gastro-enteritis from cholera infection:

W. A. D. was seen in 1898 with the following history: He suffered in 1848 from a severe attack of Asiatic cholera that was epidemic in

New Orleans during that year. His convalescence was retarded by persistent vomiting and diarrhea, which continued off and on for several months. Complete recovery finally ensued, although ever since then he has been subject to slight attacks of diarrhea during the first onset of cold weather or after glaring dietetic errors. Aside from these transient indispositions and a winter cough the result of chronic bronchitis with emphysema, he has remained well and able to eat what has pleased him, without discomfort.

When seen in 1898 he was seventy-four years of age and was found to have achylia. For the past twelve years this has continued, and he is at the present time a hale and hearty old gentleman, eighty-nine years of age, who is free from all digestive trouble and who enjoys life with keen relish.

The absence of gastric symptoms is due to two causes:

In uncomplicated achylia motor errors do not occur. The stomach shows normal, or as many believe, increased motor power, so that its contents are passed into the intestine within normal time limits to say the least. Stagnation and fermentation of food do not occur. Flatulence is not a symptom of the disease. Achylia with gastric flatulency regularly implies the existence of some complicating motor error. Achylia with stagnation suggests malignancy and justifies exploration.

Intestinal digestion is naturally capable of doing all the work required for the maintenance of health and nutrition, so that gastric digestion is in a way superfluous. Should, however, bowel disturbances occur, this compensatory digestion is rendered imperfect, malnutrition and diarrhea are apt to occur. The lack of intestinal digestion is never in achylia sufficient in itself to induce progressive loss of flesh to any great extent, unless there be diarrhea—the loss of weight when it occurs, is attributable rather to this latter condition and proportionate to its severity than to insufficient compensatory digestion within the intestinal tract. Progressive loss of flesh and strength that is not commensurate with the severity and chronicity of the diarrhea should arouse the suspicion that more than simple achylia is present.

Gastric Symptoms.—Gastric symptoms are but rarely present in uncomplicated achylia.

In a certain number of patients, 3 per cent. of the writer's cases, "heart-burn" and "acidity" occasioned considerable distress. The patient complains that about one hour after he takes his food, he is annoyed by a burning sensation in the stomach, relieved by alkalies or by eating, and his description is the same as that given by those whose gastric contents at this time are excessively acid, so that there is no way by which a differential diagnosis may be made except by means of

the stomach-tube. Withdrawal of the gastric contents at the time at which this distress is present, usually shows that the gastric contents are free from all acidity and are dryish and of a squeezed-out appearance. This abnormal dryness of the contents of the stomach is probably the reason for the subjective distress, a supposition that is further corroborated by the fact that drinking a glass of plain water will relieve as completely as if a similar quantity of alkaline fluid had been taken. The following is the history of such a case of spurious hyperacidity occurring in achylia.

H. C., aged thirty-five years, was well and without digestive ailments or distress of any kind until four years ago. At that time without any apparent cause he began to suffer from a burning pain in the stomach appearing one or two hours after meals, intensified by mental excitement or physical fatigue. Eating and drinking soda in Vichy regularly relieve his distress. Of late he has complained of a moderate amount of gas in his stomach. He has remained physically well. Chief complaint is heart-burn.

Physical examination shows a healthy looking man. The lower curvature of the stomach lies 4 cm. below the navel and is moderately atonic.

Fasting stomach empty.

Test breakfast, dry achylia; total acidity 6. Zymogen active, $\frac{1}{100}$.

In this case the flatulence of which he complained could be probably attributed to his gastropnoxis and atony, rather than to his achylia.

Nausea and vomiting occur so rarely in achylia, that their presence suggests the possibility of some complication to which they may be due. Persistent vomiting suggests malignancy, occasional attacks of vomiting with pain suggest gall-bladder complications.

Intestinal Symptoms.—The symptoms due to achylia are chiefly intestinal, and consist of (1) diarrhea; (2) intestinal flatulence and discomfort; (3) intestinal toxemia.

Diarrhea.—Diarrhea is the most frequent symptom of achylia, and was present in 34 per cent. of the writer's cases. Strauss reports that it occurred in 36 per cent. of the achylia cases that he has seen.

In many cases, merely a tendency to diarrhea exists. From time to time attacks occur described by the patients as ordinary "summer complaint," although they are usually more prolonged and show a greater tendency to recur. These outbreaks are more frequent in summer than in winter; they may follow a definite dietetic error or there may be no apparent reason for them that we can discover.

In other instances the patients will go steadily along, having one or two loose movements before or after breakfast—rarely being annoyed during the day. There may be abdominal discomfort and rumbling

preceding the evacuation, and considerable flatus may be expelled, but pain in these milder cases does not occur.

In more severe instances the diarrheal movements become more frequent and materially interfere with the nutrition and general health of the patient. The diarrhea occurs with its greatest intensity during the early morning hours, and usually becomes less marked or even ceases after the early part of the forenoon. It would seem as if food taken during the day fermented and irritated the bowel during the night, and was evacuated in the early morning. This predilection for the early morning hours is characteristic of diarrhea that owes its origin to disturbances in gastric chemistry, and is seen both in cases of hyperacidity, and in cases of achylia, although it is much more frequent in the latter condition.

Examination of the stools is often quite characteristic. In the milder cases we may find only a few undigested meat fibers and a great excess of connective tissue in threads and flakes occasionally appearing as a fine network throughout a stool that may otherwise appear normal. The presence of connective tissue in excess especially when it appears as a macroscopic residue, clearly shows a lack of gastric secretion, for we know that connective tissue unless thoroughly cooked and softened, is digested only or chiefly in the stomach.

In cases of greater severity the stools are large and liquid and usually quite offensive by an excess of fatty acids and soaps. Undigested meat fibers are usually present in considerable number and undigested connective tissue is apt to be sufficiently abundant to form a residue that is evident to the naked eye.

In a certain number of these stools evidence may be present of a catarrhal inflammation of the small intestines.

Enteritis may be due to the irritation of the mucous membrane of the small bowel by undigested food, or may arise from excessive bacterial growth that is in turn due to the presence of an excess of undigested proteid matter within the lumen of the bowel. The mechanical irritation caused by the undigested food entering the bowel may also be considered quite a potent cause.

The writer has found that in the cases of diarrhea that occur with achylia, evidences of enteric catarrh are found in the stools of three-quarters of the number. Mucus is present, freely and intimately admixed with the more solid portions of fecal matter, indicating that its origin is above the large intestine, although we are unable in many cases to state which portion of the small intestine is the more involved. If under the microscope small clusters of undigested starch granules are surrounded by a capsule of fine mucus, the catarrhal process probably involves the upper and midportions of the jejunum. Mucus with

leukocytes and intestinal epithelium that are intimately admixed also indicates catarrhal processes in the small bowel.

To determine the probable origin of intestinal mucus Krauss recommends the following method of examination.

A fecal smear is stained with a 1 per cent. solution of alizarin sodium sulphonate. Mucus that is normally present in the stools, the "cohesion mucus" as it was termed by Nothnagel, is evidenced by the appearance of small flakes and scales tinged faintly yellow. The farther the mucus has to travel or, in other words, the higher is its origin within the bowel the more faintly it is stained. Bright large red flakes have their source in the lower part of the large intestine—mucus from the smaller intestine is stained lightly yellow, and the nearer the source of the mucus is to the duodenum the paler is the tint.

In other cases there are no evidences in the stools of any catarrh of the intestine that we can discover—the evacuations are pea soup in consistency and their liquid character seems chiefly due to increased intestinal peristalsis. It is in these cases that small doses of bromides are followed by such brilliant results.

Evidences of colitis in the stools, such as the presence of large masses of mucus not intimately admixed with the fecal matter but lying free or coating large fecal masses, are not usually seen in achylia unless from some intercurrent complication.

Occult bleeding does not occur in achylia. Repeated positive blood reactions in the stools suggest the possibility of malignancy.

Abdominal Distress and Distention.—Abdominal distress and distention occur in many of the cases, usually but not necessarily associated with diarrhea. The discomfort is usually more or less constant throughout the day, or may be limited to the morning hours. So frequently do the symptoms of intestinal indigestion owe their origin to changes in gastric secretions, that in every case presenting these symptoms examination of the gastric contents should be made.

Intestinal Toxemia.—Symptoms of intestinal toxemia are present in a large number of patients with achylia, although not relatively as frequent as in hyperacidity, nor are they as common in achylia without atony as in the cases in which this complication is present.

There may be recurring attacks of "biliousness," of headaches or of mental depression. There may be various forms of skin diseases dependent for their origin upon intestinal toxemia, urticaria being by far the most common. These symptoms of intestinal poisoning are not characteristic of achylia, any more than they are of a great variety of gastric and intestinal disorders.

An interesting history is the following:

Mrs. H. H. B., aged fifty years.

Twenty-one years ago a pelvic abscess, thought to be perityphlitic,

ruptured into the bowel. This was followed by complete recovery, and she remained perfectly well with the exception of attacks of what was thought to be gallstone colic twenty years ago and again six years ago.

For the past two years she has suffered indescribable torment from chronic urticaria, the body never for a moment being free from the eruption, so that she has been unable to sleep and has become both physically and nervously exhausted. She has been under constant treatment by eminent specialists, both by external applications and by a great variety of internal medication.

She gives no history of any past or present gastric symptoms of any kind whatever. Her bowels move loosely every day from laxative medicine that has been ordered for her.

Physical Examination.—Patient much exhausted from constant itching and lack of sleep. Surface of the body covered by extensive urticarial eruption. Gall-bladder neither tender nor palpable. Position and size of stomach normal—no evidences of atony. Examination otherwise negative.

Fasting stomach, normal.

Test breakfast shows dry achylia; total acidity, 6.

Lab-zymogen, negative $\frac{1}{80}$, faintly positive $\frac{1}{40}$.

Stool examination shows connective tissue present to a macroscopical degree. Fine mucus intimately admixed. Slight excess of fatty acids and soaps—odor somewhat putrefactive.

Patient was placed on an achylia diet, and ordered oxyntin by mouth, small doses of strontium bromide, and intestinal irrigations. The eruption ceased within a few days and for the past six years there has been no return. During the last year she has successfully been operated on for acute impaction of a stone in the common duct. The association of achylia with diseases of the gall-bladder and gallstones is here quite clearly indicated.

Achylia with Complications.—Achylia is so often accompanied in its course by lesions of the gall-bladder and by gastroptosis that special description should be given of the symptoms that arise from such combinations. This seems to the writer quite important, as many symptoms due entirely to these coexisting disorders are attributed to the achylia whereas in fact they owe their existence entirely to the associated disorder. If the cases of achylia are divided into three groups, one of achylia, pure and simple, one of achylia with lesions of the gall-bladder with or without symptoms of gallstones, and one in which achylia occurs with gastroptosis, it will be readily seen that many symptoms ordinarily considered to be the result of lack of gastric secretion are present only in the groups which include the complicating lesions. It is not in achylia alone that we may apply this method of studying

our cases—the more we sort out symptoms of the diseased condition or deranged function we are investigating from the symptoms of other diseased conditions or deranged functions that may coexist, either directly, or indirectly dependent or entirely independent of each other, the simpler and the less confused becomes our knowledge of the symptoms of disease.

GALL-BLADDER AND GALLSTONE COMPLICATION. — Gall-bladder or gallstone complications occur in 22 per cent. of all achylia, and add their symptoms to those of the latter disease. It has been seen that in achylia the chief symptoms are intestinal and that gastric symptoms are insignificant and usually absent. Gall-bladder complications (and this term is used by the writer to include all forms of gall-bladder infections and inflammations with or without the formation or evidences of the presence of stones) usually give well-marked gastric symptoms, while not interfering in the slightest with any of the functions of the intestine. Cholecystitis may well be suspected in every case of achylia that is attended by gastric symptoms.

Pain is a symptom that does not occur in uncomplicated achylia, but is frequently present in the gall-bladder cases. It may be paroxysmal and lancinating, it may be dull, boring or aching, occurring usually independently of the time of taking food, although in some cases a definite and orderly sequence of time may be observed. At other times the pain is cramp-like—a pain that is always suggestive of biliary disease. Epigastric pain in achylia should suggest gall-bladder disease, chronic appendicitis, arteriosclerosis, or malignancy.

Nausea and vomiting may occur from time to time. The nausea in these cases is usually mild in degree, and does not usually prevent the patient from a full enjoyment of his meals, after he has once sat down to the table. Vomiting may be associated with attacks of epigastric pain or may occur independently. It is quite erratic in its type.

Sudden attacks of gaseous distention of the stomach may occur. The raising of brackish or bitter-tasting fluid is not infrequently observed.

The following example of achylia thus complicated may be given.

Mrs. W., aged forty-two years, admitted April 30, 1909, with the following history.

Patient has always been self-indulgent and reluctant to take exercise. Her diet has always been injudicious, and during the past few years she has grown quite stout.

Her digestion gave her no trouble until three years ago, when she began to complain of sudden attacks of painful distention of the stomach, for which she would take a great variety of carminatives and from which she would after a few hours obtain relief by the raising of wind in great quantities. After eating she would often feel as if "there was

a great hole in her stomach" quite independent of the quality or quantity of her food. From time to time she suffered from pain in the epigastrium of a dull aching character, coming "at any time" and "by spells" often accompanied by nausea and by vomiting which afforded no relief to her distress. At other times she would go for weeks eating everything without any discomfort whatever. For the past three years she has had morning diarrhea during the summer months without intermission, while during the cooler part of the year she has suffered from only occasional outbreaks lasting from a few days to several weeks at a time.

Three years ago, at the onset of her present illness, a test breakfast disclosed achylia.

Physical Examination.—Large, stout, healthy looking woman.

Lower border of the liver cannot be determined owing to the thickness of the abdominal wall.

Gall-bladder not palpable, but deep pressure over its area elicits exquisite pain, which runs through to the back.

Test breakfast shows dry achylia.

Stool examinations show characteristic findings of enteric catarrh.

Two months later there occurred a typical attack of acute cholecystitis, with pain, tenderness, fever, nausea, and vomiting, together with well-marked rigidity of the head of the right rectus muscle.

In this case the only symptom of achylia was the diarrhea; the acute gaseous distentions, pain, nausea, and vomiting were quite characteristic of chronic cholecystitis, probably with the formation of gallstones.

GASTROPTOSIS COMPLICATIONS.—Gastroptosis complicates 20 per cent. of the cases of achylia, although it has been shown that the occurrence of these two conditions is independent of each other. The only symptom that is added by the achylia to those due to the gastroptosis is diarrhea, although this is less frequently observed than in the cases in which achylia occurs alone.

In achylia without diarrhea no symptoms whatever are added to those of the gastroptosis. Loss of flesh and of strength that occur in many cases of achylia that are not the result of diarrhea may be explained by the existence of a coexisting gastroptosis and atony, otherwise we should be suspicious of malignancy. The two following examples of achylia with associated gastroptosis may be given:

Achylia with Gastroptosis, Symptoms Entirely Those of Gastroptosis.—

Mrs. W., aged forty-five years, was well until after her husband's death fifteen years ago, she was obliged to support herself by giving music lessons. She became very nervous and overworked, and soon developed the symptoms of her present complaint, which have continued with more or less severity ever since, and there is always more or

less distress from flatulency. She is distended and bloated, although it is with difficulty that she can either raise or pass wind in sufficient quantity to relieve her distress.

For the most part she has lost her appetite altogether; at times she may start her meal with relish, which, however, is replaced by a sense of satiety after she has eaten of a small quantity.

She has lost progressively in flesh and strength and has become markedly neurasthenic. Her bowels are constipated and contain long strings of mucus.

Physical Examination.—Patient is a slight delicate-looking woman, much under weight; thorax long and narrow; costal angle forty-five degrees.

Lower border of stomach 8 centimeters below the navel, splashing readily to this point. Gastric tympany extends 8 centimeters to the right of navel. Right kidney palpable two-thirds of its extent. The lower border of the liver is one inch below the costal arch in the mammary line; its edge feels normal. Gall-bladder tenderness not elicited. Marked coloptosis present.

Fasting stomach, negative.

Test breakfast: 15 cubic centimeters; dry achylia; total acidity 6.

Lab-zymogen, positive 1:180, negative 1:160.

Achylia with Gastropotosis.—*Diarrhea the only symptom due to achylia.* M. P. S., aged fifty years, has complained for the past fifteen years of morning diarrhea, which with but few exceptions has been constant. During this time she has lost in flesh and strength, and has complained of bloating of the stomach no matter what she eats. She has grown exceedingly nervous and cannot sleep, partly on account of her nervous condition and partly because of the distress. She has abdominal pain from time to time, followed by long strings of mucus in the stools.

Physical Examination.—Tall thin woman, undernourished and slightly anemic.

Costal angle 50 degrees; general characteristics of enteroptotic habitus present. Lower border of stomach 10 centimeters below navel. Right kidney freely palpable. Marked coloptosis is present.

Fasting stomach, negative.

Test breakfast: 20 cubic centimeters; dry achylia; total acidity 6.

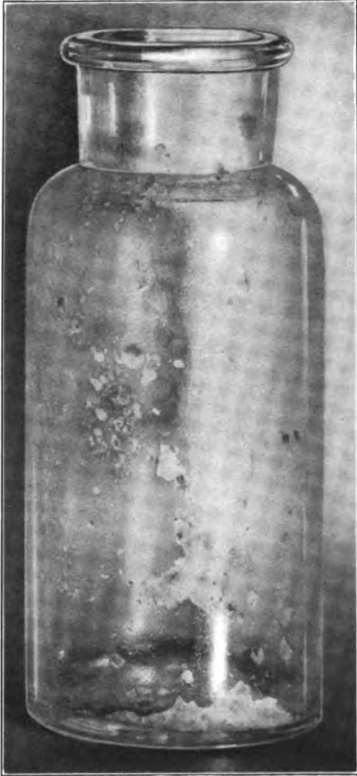
Lab-zymogen, 1:180 positive.

Lavage of the fasting stomach, negative.

Stools show the characteristic findings of a mild enteric catarrh, containing finely disseminated mucus; large masses of undigested connective tissue and many muscle fibers; quite offensive from excess of fatty acids and soaps. Strings of mucus are present, characteristic of membranous colitis.

Diagnosis.—Gastric Analysis.—The fasting stomach is almost regularly empty, although occasionally there may be aspirated small quantities of mucus or of bile-stained fluid which evidently enters the stomach from the straining efforts due to the passage of the tube. Food stagnation and bacterial overgrowth are regularly absent, as motor errors do not exist in non-malignant achylia. The absence of stagnation in any form whatever is of prime importance in differentiating simple achylia from cancer. Should any suspicion of malignancy exist repeated examina-

FIG. 103



Test breakfast in achylia. (Dry form.) Notice scanty return of undigested foodstuffs in lumps enveloped in sticky mucus adhering to the side of the bottle.

FIG. 104



A common form of test breakfast in achylia. A small amount of undigested breadstuffs is obtained, floating in a scanty quantity of clear fluid.

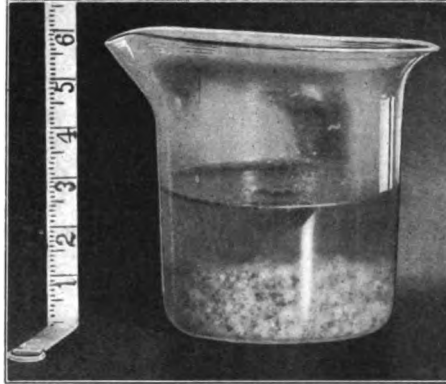
tions should be made, so that a conclusion may be arrived at without unnecessary delay. *Achylia with stagnation justifies exploration.*

Trichomonas, *megalostomas*, and various other forms of infusoria may exceptionally be present. These infusoria are not suggestive of cancer as was at one time supposed, as we now know that their breeding place is about the roots of carious teeth and that they may exist in the fasting stomach after they have been swallowed, in any case in which acid secretions in the stomach are lacking.

Test breakfast may appear in any one of three forms. The first and the most usual form is known as the *dry achylia*. The quantity extracted is small, and consists of bread fragments that look exactly as if they

had been chewed and spat out. These little fragments may be mixed with a small amount of mucus and saliva. Often only a teaspoonful of such material can be extracted, the small quantity being due rather

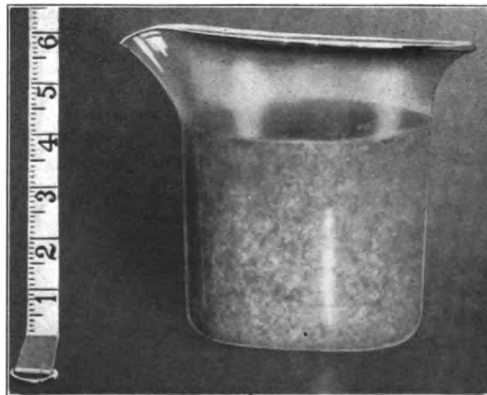
FIG. 105



Test breakfast in achylia. The undigested breadstuffs settle in a flocculent mass. The supernatant fluid is composed of very thin mucus and fluid of a viscid consistency, but not thick enough to be raised on a hook.

to the difficulty of getting such a dry material through the stomach-tube than to any hypermotility of the stomach, because if we wash out the stomach after withdrawing such a small proportion of the

FIG. 106



Test breakfast of chronic anacid gastritis. The undigested breadstuffs are seen floating in thin mucus.

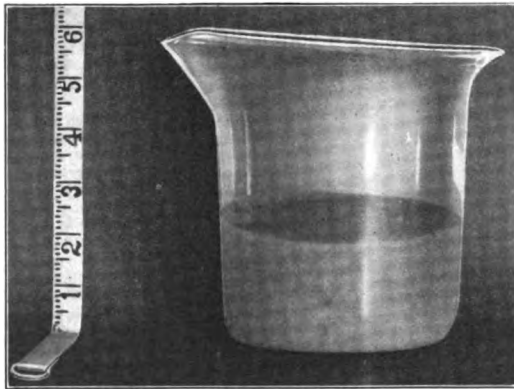
test breakfast we will find the balance in the wash-water. This form occurred in 126 out of 176 cases of achylia in the writer's series.

The second form is the "wet variety." The test breakfast consists

of a large quantity, from 30 c.c. to 200 c.c., of undigested breadstuffs, floating in a sea of mucus. This is evidently a catarrhal form, and yet in the majority of cases the stomach secretes mucus only in the digesting state, as is shown by the fact that washing of the fasting stomach does not bring out the least particle of mucus. This form occurred in 39 out of 176 cases of achylia.

The third variety is that of an apparently normal well chymified test breakfast, although on close examination it has not the purée consistency of the normal, but is of a slightly more granular appearance. This form occurred in 11 out of 176 cases of achylia.

FIG. 107



Test breakfast in achylia. A rare form in which the appearance is practically normal and well chymified.

The quantity is usually about normal, ranging from 30 to 50 c.c. and the ratio of the liquid layer to the layer of sediment is unchanged from that of the healthy test breakfast.

In rarer cases the total quantity is excessive, often from 200 to 250 c.c., and is composed largely of fluid, the depth of the supernatant fluid being three to five times the depth of the sediment. This form of test breakfast is described under alimentary hypersecretion, page 530. It would seem plausible to assume that this excessive outpouring of fluid containing no hydrochloric acid was due to an oversecretion or rather to an excessive transudation of the osmotic thinning fluid described by Strauss.

These three forms of achylia occur with the same symptoms and under the same conditions, and the writer does not know how to divide them according to any scientific classification. One form does not, however, merge into another, dry achylia remains so throughout, wet achylia remains wet achylia, the granular form does not change its appearance so as to resemble either of the other forms.

A faint biuret test is nearly always present. Starch reactions are usually carried to the maltose stage. Erythrodextrin reactions are exceedingly weak or absent.

Pepsin activity is usually reduced, although it may be that zymogens are present in full activity.

It has been attempted by estimation of the strength of zymogen in various dilutions to form some idea of the extent to which the glandular structures have been affected. The method of testing zymogen activity and the conclusions which are warranted by such tests are given on page 58.

While in a general way these conclusions may serve as a guide in our prognosis, they are not always to be relied upon, and we are not warranted in expressing ourselves too definitely as to the gravity of the case by this test alone.

Treatment.—The chief indication for treatment is to select a diet which does not call too loudly for gastric digestion, but which can undergo, by the salivary, pancreatic, and intestinal secretions the changes requisite for its proper absorption. For this purpose the carbohydrates may be given freely, fats should be slightly reduced, proteids given but sparingly.

Dietetic Treatment.—Much depends on the selection of a proper diet upon the severity of the symptoms presented. To insist too strenuously upon a diet composed chiefly of invalid foods, or of pre-digested nourishment, while perfectly suitable for one with whom constant and exhausting diarrhea, would be extremely injudicious and unnecessary for another who is free from all discomfort and in whom achylia has been found as the result of a routine examination of gastric contents. Good common-sense must be used in the selection of a diet that the patient can continue for a long period of time without aversion, and which is adequate to preserve a good nutrition and a normal weight. It will be found convenient to consider the diet: (1) in those who have no gastric or intestinal symptoms and (2) in those with diarrhea.

1. In the ordinary case of achylia without diarrhea, it is usually sufficient to eliminate all red meats, allowing fish, chicken, and fowl (except goose and domestic duck). Lamb and tender mutton may occasionally be taken, although it must be insisted that they be thoroughly cooked so that the connective-tissue framework of the meat is soft and the more easily digested by the intestinal secretions. Eggs may be given to bring the total proteid value up to the requisite.

Thorough mastication is essential both to macerate the food and bring in into close contact with the salivary ferment, and to prevent the introduction of large masses of food into the stomach and thence unchanged into the bowel. Defective teeth should receive immediate

attention. Food should be finely cut, or even minced. Vegetables should by preference be mashed by a fork, or put through a purée sieve—they should never be swallowed in large pieces. Beef broths, consommé, and peptone solutions may be given for their stimulating effect upon the gastric glands.

It is not advisable to recommend highly seasoned or overspiced food, or alcoholic “appetizers” for this purpose, as by so doing we may increase gastric inflammation, of which achylia is the evidence. Raw fruit is generally inadvisable owing to the excessive amount of cellulose. Cooked fruit may be taken freely except when there is a tendency toward morning diarrhea.

The diet must be varied so that the patient escapes the deadly monotony of eating the same food day after day, and on the other hand should be reasonably within his means. Laboring men cannot afford chickens and fresh fish. For them thoroughly cooked stews may be allowed. A sample diet is as follows:

BREAKFAST:

Cup of coffee, or cocoa, with cream or sugar. Cereal, with cream and sugar, or saccharin. Two soft-boiled or poached eggs. Rolls, toast, pulled bread, or zwiebach. Moderate amount of butter, preferably unsalted. Strained pure honey, such as Sheffield Farms. Orange marmalade or any Dundee jam. No hot bread. No fruit.

10 to 11 A.M.

Choice of: Glass of top milk or cream, or milk and cream, with crackers. Cup custard. Malted milk. Junket. Buttermilk or lactone milk. Russell's emulsion. Egg shake.

LUNCHEON:

None: Liquids restricted to less than one glass. No steak, roast beef, sweetbreads, kidneys, pork. No salt fish or shell-fish. No radishes, raw celery. No anchovy. No oranges, grape fruit, or raw apples.

Allowed: Soup, fresh fish, chicken, turkey, fowl of all kinds, save domestic duck and goose; lamb, mutton, lean broiled or boiled ham (occasionally). Simply prepared ragout. Oysters in any form. Olives and caviar allowed.

Freely: Potatoes in any form, except fried; boiled potatoes only occasionally, and then well masticated. Carrots and turnips, etc., if passed through a purée sieve. Tender-boiled onions, tender beets, oyster plant, peas, beans, spinach, rice, macaroni, spaghetti, samp, etc.

Occasional: Cauliflower, stewed celery, Brussels sprouts, asparagus tips. Bread if not too fresh. Salad with French dressing. Cheese.

Desserts: Rice pudding, farina, corn-starch, blanc-mange, prune soufflé, ice-cream, but no fruit ices, stewed figs, or prunes; baked apple with cream, etc.

4 to 5 P.M.

Same variety as 11 A.M., with the additional choice of cocoa or chocolate, with cream or sugar, or a farinaceous dessert, as on luncheon list.

DINNER:

Same variety as lunch.

10 P.M.

Same variety as 11 A.M.

While liquids are restricted at meals, water may be taken freely between meals.

2. When diarrhea occurs, showing imperfect digestion, the diet must be more strict. The writer's second and third week ulcer diet should be advised as long as diarrhea persists. If despite this stricter diet the diarrhea should continue, the patient should be confined to bed, hot applications constantly applied to the whole abdomen in the manner described in ulcer, and the diet continued. The patient should be kept in bed until he can take the more general diet given above without return of the diarrhea.

Medicinal Treatment.—The majority of patients require no medication provided that the diet is correct. Other patients are benefited by hydrochloric acid given in some form or another. The dilute acid may be given in Mxv to xx doses well diluted during or after meals, either plain, or with some one of the aromatic elixirs, such as the elixir of calisaya. The tincture of *nux vomica* or of *physostigma* should be added if atony coexist. It is doubtful if the acid is of any direct help to gastric digestion, as the quantity that can be given is so small, but empirically good results follow its use, perhaps as an excitor of the formation of secretin. There seems to be no more benefit derived from taking the acid between meals than at the table, and it is far less convenient.

Oxyntin, an acid albumin manufactured by Fairchild Bros. & Foster, is convenient and efficient. A teaspoonful of the powder may be taken either in wafer paper or placed in sandwich form between small slices of bread and butter and taken at the meals. Two grains of oxyntin are equivalent in therapeutic value to one minim of the dilute hydrochloric acid of the *Pharmacopœia*.

Gasterin and hepatin consist of the gastric juice of dogs obtained through artificial gastric fistulas. These preparations are exceedingly active as digestants, but they are expensive, have to be freshly obtained, and kept in a cool place, and it is a question whether they are enough better than the official acid to be worth the trouble.

Acidol tablets occur in two strengths. The stronger tablet represents in strength eight minims of the dilute acid, the weaker tablet but two minims. These tablets are to be dissolved in water and taken at meals. They are of special service in the dyspepsia of aged subjects.

Pepsin is now but rarely given. The majority of the preparations in the market are inert.

Pancreatin and pancreon may be given to aid intestinal digestion. They should be combined with alkalies and given at least two hours after meals.

Carotid and papoid tablets are much in vogue, although the writer has not been impressed by their value.

Secretin on the other hand seems to be of very great service as a stimulant to pancreatic activity. The tablets made by Fairchild Bros. & Foster should be given two hours after meals, either alone or combined in a capsule with a tablet of acidol. The addition of eserine gr. $\frac{1}{50}$ is of service. There is no doubt that given in this way secretion is a valuable addition to our list of remedies.

For the diarrhea, astringents should not be used—rest in bed, external moist heat, and diet usually bring speedy relief. Tricalcic orthophosphate in dram doses may be given between meals three times a day, or gr. xv of calcium lactate at like intervals. These calcium preparations can be recommended by the writer in achylia diarrheas whether the patient is sent to bed or is allowed to be up and around.

Diarrhea is so frequently aggravated by an increase in intestinal peristalsis that small doses of strontium bromide are to be recommended as a routine procedure in nearly every case.

Castor oil (℥ x) and salol (gr. x) in capsule are often of value in diminishing abdominal discomfort and unrest. Xeroform in gr. x doses three times a day may be also recommended.

Mineral waters are of service only when the achylia results from gastric catarrh or is accompanied by gall-bladder disease.

In the former case hot Kissingen (Rakoczy) before meals may be given. It seems to be of no service in dry achylia nor in the cases in which the zymogens are greatly reduced in activity.

In the gall-bladder cases, Carlsbad may be recommended, although theoretically contra-indicated in conditions of anacidity. It is to be used sparingly, however, so that its effect on the bowels is reduced to a minimum, one unformed movement daily being the limit.

Medicinal waters are contra-indicated in all cases complicated by atony or other motor error, and in cases with diarrhea.

Lavage is indicated only in those cases in which the washing of the stomach in the fasting state brings in the wash-water large quantities of mucus. The secretion of mucus in the fasting state is rather uncommon in achylia, and therefore, the number of patients in whom lavage is indicated is exceedingly small. There is no benefit to be derived from lavage in the cases whose gastric contents and test breakfasts show mucus, but in whom mucus is not present in the fasting stomach.

CHAPTER XIX

HYPERSECRETION (GASTROUCCORRHEA OR "REICHMANN'S DISEASE")

THE disorder which we call hypersecretion is distinguished from other disturbances of the stomach by the fact that gastric juice is poured out in far greater quantity than is required for the purposes of digestion. The oversecretion may occur both in the digesting and in the fasting state, so that not only is there an increased amount of gastric juice secreted with the test breakfast, but demonstrable quantities of the same acid fluid are to be found in the fasting stomach. To this form the term "continuous hypersecretion" is applied. In other cases an excessive quantity of gastric juice is secreted only when food is present in the stomach, and ceases when the stomach empties itself. This is spoken of as "alimentary" or "digestive" hypersecretion. These two forms will be separately described.

Continuous hypersecretion generally occurs as a chronic process either running a more or less uniform course, or in some instances interrupted by exacerbations often so sudden and severe that they seem to indicate an acute manifestation of disease. In other cases the disorder may unexpectedly appear without the least indication that there has previously existed any evidence of the chronic form of the derangement. Acute and chronic forms are therefore to be recognized.

ACUTE HYPERSECRETION

Acute hypersecretion may appear as a new and acute disorder, although in the majority of cases it will be found to represent an acute exacerbation of the chronic form which may or may not have been previously recognized.

Etiology.—Formerly little was known about the cause for the complaint. It was supposed to be commonest among young excitable individuals, especially those who are the victims of a neurotic inheritance. Attacks were ascribed to overexcitement and cerebral fatigue. It was said to occur frequently in learned men who gave way to attacks of anger or overindulgence in alcohol. In other instances dietetic

errors have been responsible. These theories are now generally abandoned. Acute periodic hypersecretion as a pure neurosis does not occur.

Certain attacks of gastric crisis in locomotor ataxia are supposed to be due to acute hypersecretion, and there are instances in which there seems to be an intimate connection between the two conditions, but there is little if any proof that a direct excitation of secretory nerve stimulus is the real reason for the excessive and continuous hypersecretion. It is more probable that a spasmodic condition of the pyloric sphincter is induced by the spinal disease which manifests itself in over-secretion.

Modern clinical observation illuminated by the light of surgical explorations and operations lead us to the belief that acute hypersecretion occurs only from sudden narrowing of the pyloric canal either from organic causes, such as juxtapyloric ulcer or from a muscular spasm of the pyloric sphincter. In the majority of cases a combination of organic stenosis and spasm are to be held responsible for the event. A pylorus that has been partially narrowed by the tumefaction of an acute ulcer or by the cicatrization of one that is more chronic may suddenly close down by an extension or recrudescence of the ulcerative process by tumefaction or spasm and give rise to acute hypersecretion. After the spasm has relaxed or the tumefaction subsided the acute symptoms abate, but it is rare indeed that there are not then found evidences of a milder but more chronic form of the malady, so that it would appear that the acute hypersecretion was but an acute exacerbation of a chronic disorder.

Symptoms.—As acute hypersecretion is regularly due to sudden pyloric narrowing, either organic, spasmodic, or to a combination of these two conditions, a previous history indicative of some morbid process involving directly or indirectly the patency of the pyloric orifice may generally be elicited. In the vast majority of instances the history of a preëxisting gastric or duodenal ulcer can be obtained; more rarely may there have been symptoms of a pyloric neoplasm. In reflex pylorospasm from gall-bladder or appendicular disease a previous history indicative of either of these disorders may be difficult to obtain with any clean-cut definition, and even may be entirely impossible to elicit in any form whatever.

In other cases no symptoms of any preëxisting disorder may have been observed, but the phenomena of hypersecretion appear from a clear sky, or after a trifling indigestion attributed by the patient to some d'etetic error.

The first symptoms are those of discomfort or distress. There are uneasy feelings in the epigastrium which the patient is often unable to describe with any accuracy. There may be a sense of fulness and

distention, as if the patient has overeaten, or a feeling of weakness and soreness in the back, so that the patient will attempt to straighten the back hoping to relieve the distress. In

FIG. 108



Vomited fluid of acute hypersecretion in a case of acute gastric ulcer. The vomiting of this quantity occurred thirty-five hours after total abstinence from food and drink. Measurement in centimeters.

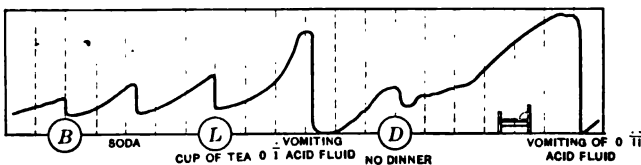
hydrochloric acid. The color may be yellowish or greenish from admixture of bile that may be regurgitated even though the pylorus

in the majority of cases *pain* is the chief complaint, is of a burning or boring character, and is quite severe and harassing. These painful or disagreeable sensations are regularly continuous, slightly relieved for the time by the taking of food or alkalies, but soon returning as severely as ever. If soda be taken in large enough doses the relief may be quite marked for the time, although gaseous distention of the stomach by the liberated CO_2 often increases the general discomfort and fulness. Complete relief is afforded only by the emptying of the stomach either by emesis or by the tube, but the period of comfort is but temporary as the symptoms return when the stomach becomes again filled with the acid fluid.

Character of Vomitus.—The character of the vomitus is pathognomonic of the condition.

1. The vomitus consists almost entirely of fluid giving reactions for free

FIG. 109



Line of pain in acute hypersecretion from a patient with acute duodenal ulcer showing influence of food, alkalies, and vomiting, on the pain.

be narrowed, or brownish from altered blood. Food remains may be present if food has recently been ingested, but usually the bulk of the vomitus is of fluid alone.

2. The vomited liquid is in greater quantity than can be accounted for by fluids recently taken, and is usually quite excessive, one or more pints being ejected "by gushes." Very characteristic is this excessive fluid vomiting when the patient has been deprived for some time of all nourishment; the patient "wonders where all the fluid comes from." Even though the patient may have been fasting for twenty-four or more hours, two or three basinsful may be collected during the night. Actual nausea is rare, the vomiting being usually preceded by a short period of fulness and uneasiness followed by effortless vomiting of the acid liquid. During the attack the patient shows rapid exhaustion and loss of weight. The pulse becomes small and thready, the body temperature depressed. Thirst is bitterly complained of, but every attempt to assuage it is followed by gastric unrest and a repetition of the vomiting.

Other Symptoms.—In some instances severe headache with photophobia, suffusion of the conjunctiva, and even temporary diplopia may be observed, closely resembling migraine. Transient delirium has been known to occur. The cephalalgia seems to depend upon the presence of the acid fluid within the stomach, for it promptly ceases whenever the stomach is emptied by lavage or emesis. To acute hypersecretion accompanied by migraine, Roszbach gave the name of "gastroxynsis." Whether the migraine causes the hypersecretion, or whether hypersecretion occurs first and is then followed by a systemic headache, cannot be dogmatically decided. The writer believes that in the vast majority of cases the migraine is merely symptomatic of the secretory disorder. In his experience typical periodical migraine with a resulting hypersecretion has not been observed. A purely neurotic form of acute periodical hypersecretion may be disregarded.

The bowels are regularly constipated. The urine is diminished in quantity and shows the characteristics of concentration. The chlorides are reduced, urea is diminished. The urine may present a peculiar glistening appearance due to the presence of numberless uric acid crystals, which according to Fenwick precede the attack and indicate the proximity of the crisis. Positive reactions for acetone and diacetic acid may frequently be obtained. During the attack the upper abdominal wall may be retracted—the epigastrium is usually diffusely tender. Gastric stiffening is rarely observed, indications of an increased peristalsis are seldom present. Splashing sound may be audible by gentle tapotement unless the abdominal wall be thick or rigid, and are quite suggestive of the derangement if they are heard when the stomach should be empty.

The attack often subsides abruptly. Occasionally the crisis is marked by gurgling noises in the stomach and by a feeling that an internal

spasm has suddenly relaxed. As soon as the paroxysm is over the patient is able to take nourishment, and within a few hours may declare that he feels as well as ever, although examination may show a considerable amount of acid fluid still remaining in the stomach, indicating that a mild form of hypersecretion still exists without, however, giving rise to noticeable symptoms.

Duration and Course.—The duration of the attack varies from a few hours to several days in the ordinary cases; the subsidence may be complete or the condition may gradually merge into that of the chronic form. During the course of chronic ulcer with chronic hypersecretion, acute exacerbations may occur of great severity and may last without remission until the patient is relieved by operation or dies exhausted.

The varieties in the course of acute hypersecretion are fully described under the heading of ulcer.

Diagnosis.—No difficulty whatever should arise when a patient begins to vomit large quantities of acid fluid and continues to do so after the stomach has once been emptied. To call such cases acute indigestion or acute gastritis implies culpable ignorance or neglect. Difficulty may be experienced, however, in recognizing this ailment before the event of vomiting. It is always suggestive when a patient, especially if there be an ulcer history, complains of a constant epigastric distress relieved slightly and temporarily, if at all, by eating or by alkalis in average doses. The passage of a stomach-tube will at once reveal the cause for the suffering.

Acute hypersecretion is, however, merely a symptom-complex of pyloric obstruction in one form or another, and the diagnosis is incomplete unless we are able to discover the underlying cause for the sudden loss of patency. The previous history should be most carefully taken, especially in reference to possible ulcer or cancer, and to attacks of gall-bladder or appendicular disease. The absence of any data indicating the previous existence of any of these disorders does not necessarily exclude them as possibilities. In children reflex pylorospasm from inflamed and adherent prepuce may occasion recurring outbreaks of excessive gastric secretion.

In every case the gastric crisis of tabes must be considered as a possibility, unless it can be excluded by the absence of characteristic symptoms and physical signs, remembering, however, that attacks of gastric crisis may occur during the preataxic stage of the spinal disease. In gastric crisis the vomited matters are not always hyperacid, they may be subacid or even alkaline, and the emesis is not followed by the same tranquillity that occurs with acute hypersecretion.

It is of importance to discriminate, if possible, between pyloric spasm and organic stenosis. The two conditions are often combined

so that separation of the pyloric narrowing into two component parts is naturally difficult. The more sudden the attack and the shorter its duration the greater the probability of spasm. Hypersecretion from reflex pylorospasm due to appendix or gall-bladder disease is rarely severe and seldom continues longer than twelve to twenty-four hours. Attacks which last more than two days are almost certainly due to organic stenosis with or without a concomitant spasm. In attacks lasting four or five days an organic cause may be considered certain, and the spasm element practically negligible. The degree of permanent constriction of the pylorus can best be determined by a series of gastric analyses made after the acute manifestations of the malady have subsided.

Illustrative Cases.—*Acute Hypersecretion Attacks Due to Chronic Ulcer.*—Mrs. H., aged thirty-five years, was well until one year ago, when she began to suffer from attacks of gnawing pain in the stomach relieved only slightly by eating or taking soda, but completely relieved for the time being by vomiting. During such an attack she would vomit whether she ate or not, the vomited matters consisting of acid water which would come up “by great gulps.” These attacks would continue twenty-four to forty-eight hours and then suddenly subside, leaving her quite well in the intervals, until two months ago, when she began to complain of more constant distress and heart-burn appearing about two hours after meals totally relieved by eating. Six weeks ago she began one of her periodical acute attacks in which she vomited acid water for fourteen days, although she took very little food. Five days ago another attack began and since then she has vomited from 2 to 4 pints daily of acid fluid (total acidity 104, free hydrochloric acid 86), although the total amount of liquid she has been able to take has not exceeded 10 ounces a day.

Operation shows chronic ulcer of the lesser curvature encroaching upon the pyloric canal.

Acute Hypersecretion with Cancer.—J. S. M., aged forty-seven years, was a healthy man until six weeks ago, when he began to complain of burning distress in the stomach unrelieved by eating or alkalies, and from time to time vomiting large quantities of a dark brown acid fluid. The day before admission he awoke with distress in the stomach, and before breakfast there were withdrawn by the tube 3 pints of acid fluid, total acidity 98, free hydrochloric acid 64. Nothing was given by mouth until the following morning at 8 o'clock, when he vomited 2 quarts of acid brown fluid and one and a half hours later an equal quantity of the same fluid was removed by the tube, making 4 quarts in all that were withdrawn from his fasting stomach. Total acidity 106, free hydrochloric acid 80. He was operated on the following morning and a carcinoma of the pylorus with infiltration of the gastrocolic omentum was found.

Acute Hypersecretion with Chronic Appendicitis.—S. J., aged forty-five years, for a number of years has complained of acidity and heart-burn, appearing usually but not invariably at a definite time after eating, but always relieved by soda. During the past two years he has had attacks of a burning distress which would appear during the forenoon and increase during the day, not relieved by eating and but slightly by soda. The distress would prevent his sleeping until about one or two o'clock in the morning. He would then vomit his dinner, together with copious quantities of very acid fluid and then be comfortable, sleep well the remainder of the night and awake as usual in the morning. Operation showed a chronic obliterated appendix which was removed. No ulcer could be demonstrated, gall-bladder normal. Since his operation he has not been troubled by any of his former complaints.

Acute Hypersecretion Due to Adherent Prepuce.—C. P., aged twelve years, had never had any trouble with his stomach until the past few years, during which time he has had a number of attacks of persistent vomiting of acid fluid, lasting twenty-four to thirty-six hours. The termination of each attack was abrupt, and in the interval he was free from all the symptoms. His attacks ceased altogether after operation for elongated and adherent prepuce.

Prognosis.—The prognosis depends largely upon the cause. The longer the attack and the more copious the hypersecretion, the greater is the liability of there being an organic cause which requires surgical consideration, and the less the likelihood of a simple spasm. The danger of hemorrhage either from exacerbation of a preëxisting ulcer or from hemorrhagic erosions that result from prolonged and violent pyloric spasm must always be considered. In severe and prolonged attacks tetany may occur.

Treatment.—The first indication for treatment is to relieve pyloric spasm, hoping to cut short the attack and to minimize the tendency toward erosions and hemorrhages. All food and drink should be absolutely interdicted and the stomach should be emptied either by emesis or preferably through the tube. Alkalies should be given in sufficient doses to neutralize the acid fluid that may be in the stomach. External heat is not only of service in reducing the spasm but also in alleviating the distress. Atropine is a useful drug pushed to mild physiological limits, but it increases the thirst that is in itself intolerable. Morphine should not be used unless the pain be so great and unrelieved by other forms of medication that something has to be done to alleviate the distress.

In every case there arises the question of operation. If acute ulcer be the probable cause it is better not to operate, as in all probability the attack will subside and the ulcer will heal under medical treatment

alone. In chronic ulcer the disease is one which requires the soundest judgment. If we knew that the attack would be of short duration our advice would naturally be to wait until the acute symptoms had subsided. The danger lies in waiting too long so that the operation is performed upon a patient exhausted by repeated vomiting, whose tissues are desiccated by the rapid withdrawal of water from the system. Experience shows that these patients are not good subjects for operation. It may be said, as a general rule, that if the attack continues after forty-eight hours, in spite of medical treatment, immediate operation must be considered. It is important, however, to introduce fluids into the system before the operation, either by enemas or the Murphy drip, or if the case be urgent, by hypodermoclysis.

The above rules for treatment apply also to the cases that are due to cancer of the pylorus. In hypersecretion accompanying gall-bladder or appendicular disease the question of immediate operation seldom if ever is forced upon us, as the attack is regularly short. After the attack is over there may arise the question of operating for the relief of the primary disease. Each case must then be decided upon its own merits.

CHRONIC HYPERSECRETION

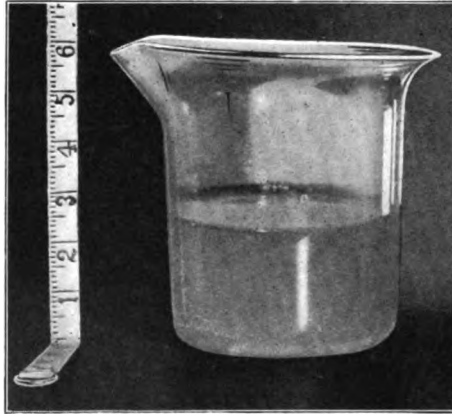
Chronic hypersecretion is characterized by the constant presence of gastric juice in the fasting stomach. Before this diagnostic rule can be applied it must be assumed that the normal fasting stomach is empty, or practically so, and this fact is universally acceded. Small quantities of fluid giving acid reactions may occur in the normal stomach but not usually exceeding 10 c.c. in amount. Larger quantities, from 20 to 30 c.c., may occasionally be found in otherwise healthy stomachs as a temporary occurrence, but such a hypersecretion cannot usually be demonstrated on subsequent examinations. The idea has been absolutely abandoned that overstimulation of the gastric glands may result from the mechanical irritation of a stomach-tube.

The fluid must be pretty constantly found in the fasting state for its presence to possess much diagnostic value. The amount of fasting fluid that is required to establish the diagnosis is an arbitrary one, variously estimated at from 20 to 100 c.c. It is, however, generally admitted that quantities over 20 to 30 c.c. if fairly constantly present, are sufficient evidences of the disorder, and the writer regards 30 c.c. as the limit past which a pathological condition begins, provided that this amount be repeatedly found. *Chronic hypersecretion cannot be diagnosed with any certainty by one examination alone.*

Two characteristics of the fluid are essential:

1. Reactions for free hydrochloric acid must be present. It was formerly regarded essential that maltose, erythrodextrin, and proteid

FIG. 110



Chronic hypersecretion. Fasting contents.

FIG. 111



Test breakfast in chronic hypersecretion, showing supernatant excessive gastric juice.

reactions should not be present, thus separating secretions from retentions, but this distinction is not insisted upon at the present time.

2. In simple hypersecretion, gross and recognizable food remains should not be present in the fasting fluid. Their occurrence takes the condition out of the hypersecretion group and places it in that of pyloric stenosis—a difference only perhaps in degree, but useful for clinical purposes. Microscopical food remains are often, however, present, consisting chiefly of undigested starch granules and finely divided carbohydrate residue, but the quantity is so small and the food fragments so minute that they are not recognizable as such by the naked eye.

Frequency.—The frequency of the disorder depends on the amount of fasting secretion that is considered necessary for diagnosis. Boas considers 100 c.c. essential, although less than this amount may be considered pathological if the clinical symptoms of acidity and pain be present. Reichmann, who took this view, that to us appears to be extreme, met with but 6 cases in several years. Friedenwald who has adopted these requirements has encountered the ailment in but 10 out of 1592 patients examined (0.65 per cent.).

To insist upon so large an amount of fluid being present seems to the writer to tend to the exclusion of positive although minor degrees of the ailment, which in justice to the patient should be diagnosticated and treated. Nor does it seem necessary to insist upon the presence of subjective symptoms, as so many and varied disorders run at times a symptomless course, even though demonstrable evidence of disease be present. Endocarditis with well-marked physical signs may be safely diagnosticated as such, although dyspnea and edema in a given case may be absent. Patients vary in their susceptibility to organic disease, some suffering from a disorder that in others is totally disregarded. It seems better to rely for diagnosis, therefore, upon demonstrable evidence of hypersecretion rather than to require corroboration by the clinical symptoms of pain and distress. The writer's opinion on this subject is held by Fenwick, who, claiming that quantities over 20 c.c. are sufficient to establish the diagnosis, considers the complaint not at all infrequent. The author, regarding 30 c.c. or over if fairly constantly present, to be indicative of pathological conditions, has found the disorder in 3.9 per cent. of private patients suffering from digestive disorders, the identical estimate of Barclay¹ (3.8 per cent.), who includes among his cases those in which the fasting secretion was 30 c.c. or over.

Etiology.—After Reichmann's classical paper in 1882 describing the symptom-complex, the view was held that the secretory excess was due primarily to overstimulation of the secretory nerve apparatus, often the result of a pure neurosis. Hypersecretion was described under the heading of "secretory neurosis." Gradually as clinical

¹ New York State Jour. Med., July, 1912, xii, No. 7.

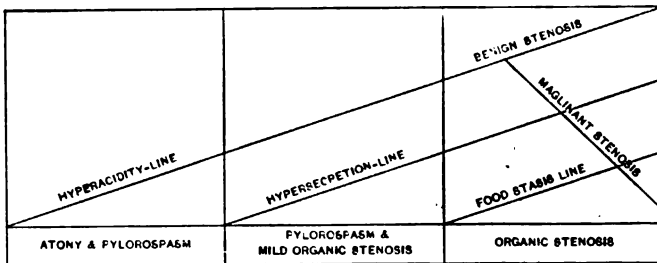
experience accumulated, it was found that motor insufficiency could be demonstrated in many instances, and that in the majority of cases ulcer or other organic lesion of the stomach was present, so that little by little the theory of neurotic overstimulation has been abandoned, although a few, including Friedenwald, of Baltimore (*Osler's Practice of Medicine*), still claim that great mental anxiety or overexcitement may be provocative of the disorder.

Motor Error.—The writer has no hesitation in stating that the one and only one cause for hypersecretion is a motor error dependent upon spasmodic or organic stenosis of the pylorus and that the degree to which fasting hypersecretion occurs, affords a reliable estimate of the extent to which the pylorus has temporarily or permanently lost its patency. The writer's conclusions from a study of his cases of hyperacidity and hypersecretion are as follows:

There are three grades of motor insufficiency.

In the first or mildest grade are included atony and slight degrees of pyloric contraction. This group is characterized by a hyperacidity which increases in direct proportion to the extent to which the motor error is developed. Hypersecretion does not occur in the cases included in this group.

FIG. 112



A second group comprises cases in which there is a slight or moderate degree of pyloric stenosis, either of spasmodic or of organic origin. This group is characterized by an increased hyperacidity and an increased hypersecretion, both proportionate to the degree of the stenosis. Atony is not included as a cause among these cases, as the motor error induced by it is not sufficient to cause a fasting hypersecretion.

The third group includes instances of well-marked pyloric stenosis, usually organic, occasionally complicated by pylorospasm, in which hypersecretion is combined by the finding of coarse recognizable food remains in the fasting state. Hyperacidity is observed on an ascending scale as the pyloric lesion increases in the benign cases, and on a descending scale in those instances in which the lesion in the pylorus is malignant.

Rules without exception are rare, but the writer believes that the above classification will be generally found true in actual experience. These three groups and their characteristic secretory disturbance are graphically illustrated in Fig. 112.

CAUSES OF MOTOR ERROR.—The actual cause for the motor error will generally be found to be:

1. Ulcer in the vicinity of the pylorus either on the gastric or on the duodenal side.

2. Carcinoma interfering with the patency of the pyloric canal.

3. Reflex spasm from gallstones or chronic appendicitis.

A protective spasm of the pylorus may occur as a reflex result from any irritative lesion in the midgut or its derivatives, best illustrated in gallstones and appendicitis, but other lesions, such as pancreatic calculus, cecal tuberculosis or cancer of the appendix, may produce identical results.

Pyloric Ulcer.—With ulcer, pyloric implantation seems to be almost always essential for hypersecretion, although in rare instances ulcer at the cardia or fundus may occasion similar secretory excess.

Fenwick observed that one of the most severe clinical examples of hypersecretion was associated with ulcer near the cardia. This localization does not, however, exclude the possibility of a grave motor error either by adhesions or by infiltration and thickening of the stomach wall sufficient to block peristaltic waves.

Amounts of fasting fluid from 20 to 50 c.c. were found in 50 per cent. of the writer's cases of ulcer, while in his hypersecretions, gastric or duodenal ulcer could be positively diagnosed in 18 per cent. of the cases.

Pyloric Cancer.—Hypersecretion with pyloric cancer is more frequent than ordinarily supposed, especially when malignancy invades the walls and base of a preëxisting ulcer. In 18 per cent. of the writer's series of cancer, 50 c.c. or more of a clear fluid giving hydrochloric acid reactions were found in the fasting state, and in his series of hypersecretions cancer could be demonstrated in 6 per cent. of the cases.

Pyloric Spasm Due to Gallstones.—Pyloric spasm due to gallstones seems to be most common when one or more stones are present in the gall-bladder too large to engage in the common duct, so that a previous history of colic or jaundice is rarely elicited. In the writer's series of hypersecretions, 6 per cent. were due to the demonstrable presence of gallstones.

Pylorospasm and Chronic Appendicitis.—Pylorospasm is a common complication of chronic appendicitis that is usually otherwise latent, and it is interesting, as it explains many of the cases of hypersecretion

in which no lesion of the stomach or duodenum can be found at operation. Before this association was commonly known, many gastrojejunostomies were done without relief to the patient, the after-history clearly demonstrating that a chronic appendicitis was the sole cause for the original complaint. The writer cannot do better than quote Fenwick¹ in this connection:

"Until the year 1907 I had felt convinced from postmortem evidence as well as from the more limited results afforded by operations that 88 per cent. of all cases of chronic hypersecretion were accompanied by a demonstrable lesion of the digestive organs, while in the remaining 12 per cent. no disease that appeared to have any connection with the stomach could constantly be detected. I was, however, well acquainted with a peculiar type of hypersecretion in which death frequently occurred from appendicitis, and was in the habit of warning such patients of their special liability to that disease; but it was not until an opportunity occurred in that year of discussing the subject with W. J. Mayo, of Rochester, Minn., that the cause of this appendicitis and also an explanation of the 12 per cent. of cases hitherto unaccounted for at once became apparent. That distinguished surgeon informed me that he had often discovered latent disease of the appendix in persons who seemed to require gastrojejunostomy, and that the removal of the appendix was followed by the subsidence of the gastric symptoms provided that the alimentary tract was otherwise healthy. Furthermore, that several of his earlier cases of gastrojejunostomy which had so materially benefited by the operation had subsequently been found to possess a diseased appendix and that when this had been removed a cure had resulted. With these facts in mind, the various surgeons who have operated for me on cases of chronic hypersecretion during the last two years have examined the appendix as well as the other important abdominal viscera, and the results obtained in 112 consecutive cases are as follows:

Chronic ulcer of the stomach existed alone in	13 cases
Chronic duodenal ulcer existed alone in	46 cases
Gallstones existed alone in	12 cases
Disease of the appendix existed alone in	22 cases
Gastric and duodenal ulcers coexisted in	3 cases
Duodenal ulcer and gallstones coexisted in	3 cases
Gastric ulcer and diseased appendix coexisted in	5 cases
Duodenal ulcer and diseased appendix coexisted in	4 cases
Cancer of the pylorus existed alone in	4 cases
	<hr/> 112 cases

¹ Lancet, March 12, 1912.

The dependence of hypersecretion upon an organic lesion of the digestive organs appears to be further corroborated by the results of treatment. Thus in almost every case where gastrojejunostomy was performed for gastric or duodenal ulceration, and the gall-bladder and appendix were proved to be healthy, the symptoms of disordered digestion gradually disappeared, and in those instances that were carefully examined subsequently by Paterson the hypersecretion was found to have absolutely ceased. In like manner the removal of gallstones when other parts of the digestive tract were healthy was followed by a subsidence of the secretory disorder, while appendectomy, after an initial rise in the percentage of the hydrochloric acid, was usually attended by similar results, although convalescence was sometimes unduly prolonged.

It is difficult to estimate with any degree of accuracy the percentage of hypersecretions that are due to chronic appendicitis, owing to the latency of its clinical course and the absence of definite physical signs. It is probable that repeated examinations might in time indicate the appendicular origin of the disorder, but many patients pass from observation before it is possible to arrive at any definite conclusion. In the writer's series of hypersecretions, 18 per cent. were due to appendicitis that could be diagnosticated with more or less certainty, while in an equal number of patients an appendicular origin could be suspected but could not be proved. Chronic appendicitis is probably the most common cause for hypersecretion of mild degree, and ulcer for hypersecretion of excessive degree.

Pathology.—Very little is known of the cellular changes in chronic hypersecretion. Degeneration of the chief cells with normal preservation of the border cells has been observed, together with interstitial inflammation in varying stages of development. It is not known whether these lesions are the cause or the result of the excessive secretion. In many of the cases examined by Korcynski and Jaworski no deviations from the normal could be detected.

Symptoms.—The symptoms are those common to hyperacidity, heart-burn, and distress, but are characteristic in the time of their appearance in relation to the meals, disappearing for several hours after a meal is taken, depending upon the quality and the quantity of the food that has been eaten, and then reappearing after the free acid is no longer neutralized or combined with the food, to last until the patient eats again. In some instances the symptoms may appear only during the latter part of the night, while others suffer regularly between each meal. Distress before breakfast is characteristic of the disorder. Relief to the distress is regularly obtained by eating, by alkalies, and

by lavage. The relation of the symptoms to the time of eating may be illustrated by the accompanying diagram:

FIG. 113

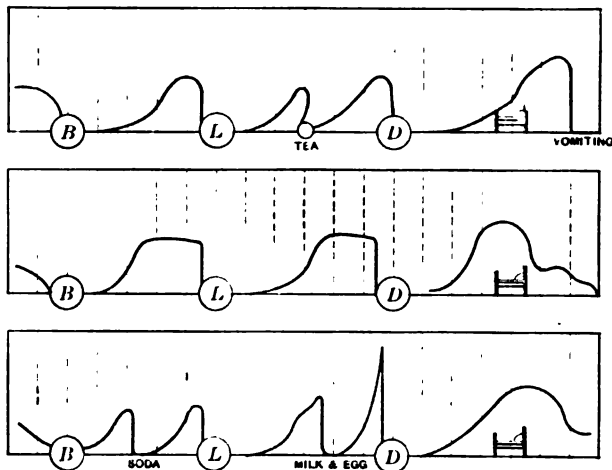


Diagram showing time line of pain and the effect on it by eating, soda, and vomiting—a three days' record of a case of chronic hypersecretion complicating duodenal ulcer.

Vomiting is not common except during the acute exacerbations of the disorder. The ejecta consist of food recently ingested, together with copious quantities of fluid acid so as to scald the throat and set the teeth on edge. The longer after meals that the vomiting occurs the fewer are the food remains and the greater the relative amount of the acid fluid.

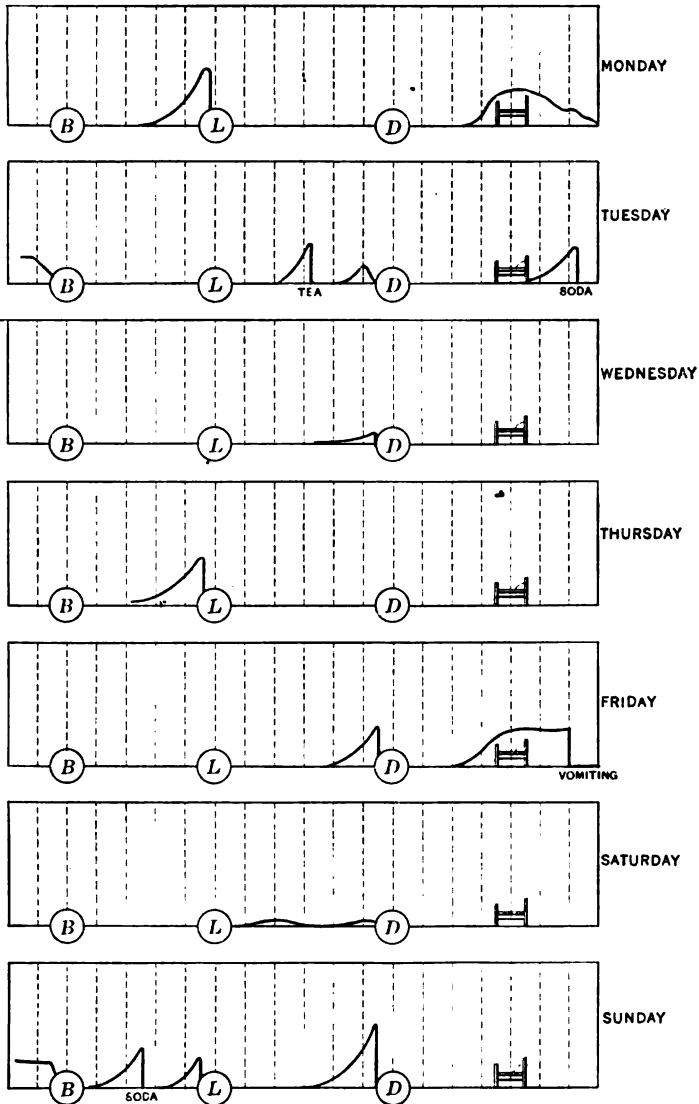
The long-continued immersion of the mucosa in an abnormally acid secretion tends to excite a congestion of the mucous membrane accompanied by interstitial hemorrhages and erosions. In the case of erosions in the pyloric portion of the stomach, traumatism by prolonged and violent pylorospasm may play an important part. These ulcers may give rise to small repeated hemorrhages or to profuse fatal hematemesis, even in the cases of ulceration which are too minute to be visible to the naked eye. It is evident that the symptoms of ulcer, except those of perforation, are due to concomitant hypersecretion.

Diminished acidity of the urine with phosphaturia may occur, in some cases apparently dependent upon the hypersecretion, but in the writer's experience most commonly resulting from large amounts of alkalies which the patient takes for his distress.

The bowels are usually constipated, although attacks of diarrhea may occur from time to time.

The extent of the hypersecretion and the severity of the distress which it produces are subject to remarkable fluctuations. Under judicious

FIG. 114

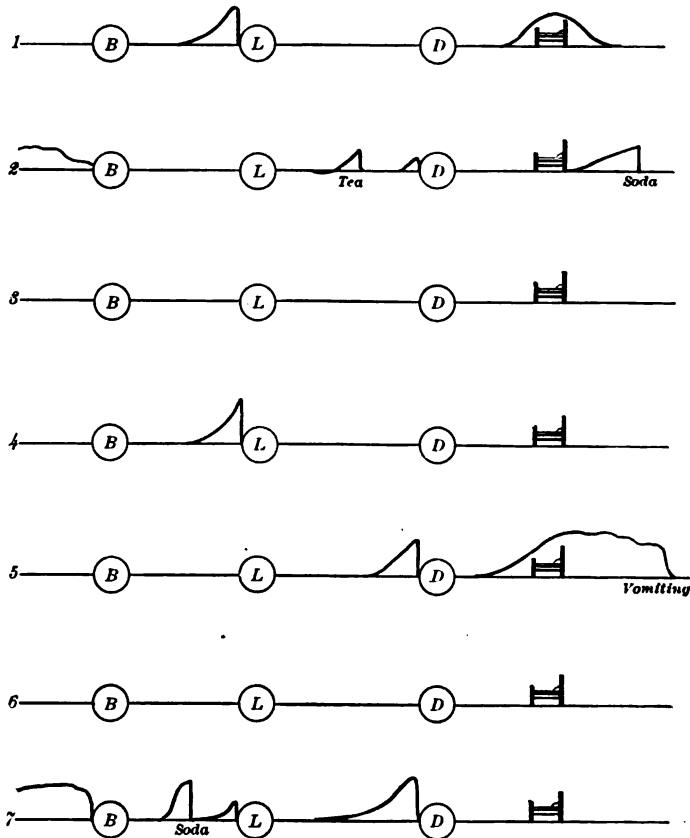


Pain chart of hypersecretion from chronic appendicitis.

treatment, and often without any treatment whatever, the symptoms may ameliorate and the secretion diminish so that at times the fasting stomach may be empty or contain a very inconsiderable quantity of

fluid. These remissions may last for a number of weeks at a time, during which there may be only occasional distress. The majority of observant patients will, however, say that they do not remember having been totally free from discomfort at some time or another in the twenty-four hours for more than two or three days at a time. The diagram (Fig. 114) shows the pain and distress record of a quiescent case of hypersecretion continued over a full week. The writer would recommend case records to be kept by such graphic method as this.

FIG. 115



Pain chart of a case of chronic hypersecretion due to duodenal ulcer, continued for one week, showing but few completely free days.

Exacerbations may occur either by dietetic error, by excessive physical or mental fatigue and excitement or without any apparent cause. In some instances the exacerbations are unusually severe and alarming, the symptoms being described under the heading of "acute hyper-

secretion." Some of these severe exacerbations subside within a day or so under appropriate treatment, while others continue with increasing severity until the death of the patient, unless relieved by a judicious operation.

Diagnosis.—Gastric Analysis.—Fasting Stomach.—The examination may be positively made by the finding in the fasting stomach of a watery fluid without admixture of gross food remains, that gives reaction for free hydrochloric acid. In mild cases proteid, maltose, and erythro-dextrin reactions are absent, but in more marked instances of the disorder in which microscopical quantities of carbohydrate food residue is found, these reactions may be present. The quantity varies from 30 to 100 c.c., rarely exceeding this latter amount unless accompanied by gross and visible evidences of food stagnation, or unless the examination be made during an acute exacerbation of the disorder.

It has been recommended that the stomach be thoroughly washed the night before the examination so as to make it sure that any fluid in the fasting state the following morning represents a secretion and not a retention. The writer does not regard this procedure at all necessary, for the test is the same whether preliminary lavage is performed or not. A meat sandwich and a glass of water should, however, always be taken on the night before the examination and thereafter nothing, not even water is to be allowed, until the tube is passed the following morning between 8 and 9 o'clock.

Test Breakfast.—Examination of the test breakfast is quite subordinate to that of the fasting stomach. The quantity abstracted is usually greater than ordinarily obtained and separates into two layers, the supernatant layer being twice or more the depth of that of the layer of sediment. This alimentary hypersecretion does not, however, always occur, as the writer has found in many instances a normal test breakfast even though 30 or more c.c. of acid fluid had been present in the fasting state. The acidity is usually higher than normal, ranging from 70 to 110, the greater part being due to free acid, the proportion between the free acid and the total acidity being usually greater than 3 or 4. The meat digestion is usually good, as shown by an excessive proteid reaction. The digestion of starches is, however, poor, maltose reactions are weak or absent, while those due to erythrodextrin and amidulin are usually marked. In some instances of extreme acidity blue reactions are obtained by the addition of the iodine water to the filtrate. The peptic power is usually greater than normal as demonstrated by the Mett or the Hammerschlag method of determination. Sarcinæ are not present unless food stagnation exists.

Differential Diagnosis.—The diagnosis of hypersecretion irrespective of its cause is a very simple matter and can be made with certainty

by the examination of the fasting stomach. The importance of repeated examinations to establish the diagnosis should be, however, emphasized. *Hypersecretion is but a symptom and the diagnosis is not complete until the underlying cause for the ailment is discovered.* Severer types of the disorder are usually indicative of ulcer, or of a benign neoplasm at the pyloric end of the stomach. Differential diagnosis between these two conditions in the absence of definite physical signs may be quite impossible. Hypersecretion due to chronic appendicitis is ordinarily of a milder form and less influenced by errors in diet. The gastric symptoms and the amount of hypersecretion show remarkable fluctuations. During exacerbation of the ailment the pain may radiate downward to the middle or lower abdominal zones, an occurrence which does not take place with ulcer. Continuous dull ache or discomfort in the epigastrium lasting several days at a time, and not due to an increase in the hypersecretion, points toward the appendicular origin of the disease.

Biliary hypersecretion consequent upon gallstones or gall-bladder disease is often characterized by periods of complete intermission. When the attacks occur, however, they are more apt to be prolonged, extending over weeks or even months. The pain may radiate to the right hypochondrium or to the right side of the back, suggesting a biliary origin for the complaint.

Vomiting is rare but acid regurgitations are especially common. Repeated examinations usually reveal tenderness of the gall-bladder. Complaint of pyrosis may be made from time to time, but this symptom is not characteristic except when it occurs at a time when the stomach is empty.

Prognosis.—The prognosis is that of the primary cause. The symptoms and the amount of hypersecretion may be modified by judicious treatment, but until the radical cause be removed recurrence may be expected.

Treatment.—The main treatment is that of the exciting cause. Cases presenting well-marked symptoms should be treated as ulcer. If the symptoms do not improve by two weeks' trial of the von Leube treatment there is very little use in continuing the cure. The question of operation should then arise. Ulcers accompanied by hypersecretion yield less readily than if the hyperacid condition did not exist, so that operative interference is more commonly indicated in these cases. Fortunately, these ulcers being usually juxtapyloric, are very suitable cases for surgical relief. No operation, however, is complete unless the conditions of the gall-bladder and especially of the appendix be investigated.

Hypersecretion depending on cancer should be investigated surgically without delay, in the hope that a radical operation may be possible.

The immediate result of gastrojejunostomy in these cases is almost always good, although the operative risk is greater than the similar operation done for ulcer.

Hypersecretion due to gall-bladder disease should be treated systematically for at least three months by medical means before the thought of operation is entertained, provided, of course, that gall-bladder infections and obstruction of the bile ducts by the stone do not occur. A prolonged course of Carlsbad water, or small repeated doses of sodium salicylate with urotropin, taken in water as hot as can be sipped, before each meal, may be advised. Rest in bed with hot applications over the liver will often be found serviceable. If at the end of two or three months the symptoms continue, the question of operation should then be brought up and surgical advice requested.

Hypersecretion due to chronic appendicitis should be treated by operation only, unless positive contra-indications exist.

Pending more radical treatment attempts should be made to reduce the amount of hypersecretion and to relieve the symptoms.

Lavage though chiefly indicated when stagnation is present often affords considerable relief and should therefore be tried. Plain water may be used or a dram of sodium bicarbonate may be added to the pint. The writer has employed with benefit solutions of artificial Vichy. Unless lavage prove beneficial within two or three weeks, there is little use in continuing it after this time unless examinations of the fasting stomach show a diminution in the amount of secreted fluid or the appearance of residual food remains.

In some cases a sufficient relief may be obtained by passing the tube in the morning before breakfast and simply aspirating the contents of the fasting stomach. A similar aspiration may be performed just before dinner. This removal of the acid fluid by aspiration alone offers no advantage over simple lavage.

The washing of the stomach with 1 to 3000 solution of nitrate of silver often proves of service.

The olive oil treatment commonly employed with pyloric stenosis may be of service in the cases supposedly complicated by pylorospasm. One or two tablespoonfuls may be given before each meal or one or two ounces may be taken at bedtime. The washing of the stomach and the introduction through the tube of large quantities of oil, as has been recommended, seems to be carrying the oil treatment too far, as it destroys the appetite and causes nausea and a repugnance for food.

The use of alkalies as a symptomatic treatment is almost inevitable. Any of the alkaline powders ordinarily prescribed may be employed for the purpose, but sufficiently large doses should be given to neutralize the fluid to the extent of relieving the pain and distress.

The use of pancreon in five-grain doses between the meals has seemed to the writer of distinct service in improving faulty starch digestion.

As hypersecretion represents a motor error, the overloading of the stomach by large quantities of alkalies and mineral waters is to be avoided.

The diet to be advised depends upon the primary cause for the hypersecretion. If ulcer be suspected an ulcer diet is to be employed, the writer's preference being decidedly toward the von Leube rather than the Lenhartz dietetic treatment.

In other cases the diet should be constructed upon the following principles: Frequent meals are always advisable so that at no time does the acid fluid lie unneutralized within the stomach. At least five meals a day should be taken. Starch digestion is usually interfered with, so that this class of foodstuffs should be diminished and an increase in the fats, such as cream and unsalted butter, should compensate for the caloric loss.

The stimulating effect of beef juice and bouillon upon the gastric digestion should be borne in mind and soups containing meat stock and peptones should be interdicted. The red beefs, such as steaks and chops, should be cut from the diet owing to the large amount of connective-tissue framework which they contain and their place in the dietary filled by fish, chicken, or fowl of any kind except goose, and ham lean but not too salty.

ALIMENTARY HYPERSECRETION

Alimentary or digestive hypersecretion is a comparatively rare form of disorder characterized by an excessive quantity of gastric juice secreted by the stimulation of food within the stomach, the phenomenon ceasing when the contents pass into the duodenum, so that the fasting stomach is empty. It is evident, therefore, that the examination of the fasting stomach should differentiate this condition from that of chronic hypersecretion and pyloric stenosis, in which demonstrable quantities of gastric juice are found in the fasting state.

Etiology.—A certain degree of alimentary hypersecretion commonly occurs with atony and gastropnoia. In these examples of atonic error the fasting stomach is empty or practically so, while the test breakfast is usually somewhat more abundant than normal and separates on standing into two layers, the uppermost liquid layer being more than equal to but never exceeding twice the depth of the sedimentary deposit. These cases while conforming to the definition of alimentary hypersecretion are not included when the term is used in a clinical sense.

The term "alimentary hypersecretion" is applied only to those cases of secretory excess in which the aspirated test breakfast is abnormally abundant, of this watery character, and which separates on standing into layers, the liquid layer being three or more times the depth of the sedimentary.

The nature of the ailment is not understood at the present time. Zweig, who with Calvo made one of the earliest contributions to the subject, regards it as due to abnormal irritation of the gastric cells not dependent upon motor error. According to this observer the disorder represents a typical secretion neurosis. In this view Zweig is supported by Elsner and Boas. Elsner considers the disorder to be a nervous disturbance of secretion, which is almost exclusively confined to patients with visceral ptoses and who present the stigmas of the enteroptotic habitus. Boas regards alimentary hypersecretion as an anomaly which may occur as a primary neurosis without any concomitant motor error. A somewhat different conception of the disorder is held by Strauss, to whom we are indebted for the first clinical description of the disease. He regards it probable that a primary hyperacidity exists which interferes with starch digestion to such an extent that undigested starch remains are retained longer than normal in the stomach and produce an increased secretory stimulation of the gastric glands. Following this acid outpouring from the gastric tubules occur the transudation of a neutral or alkaline fluid which neutralizes in part the acid gastric juice and increases still more the quantity of the liquid contents of the stomach. If the "thinning fluid" be unusually abundant the resulting admixture may be of normal or even of diminished acidity.

Whether Strauss is right in assuming that a retarded starch digestion is essential for the production of the ailment is a doubtful point, as Zweig and Calvo have demonstrated that alimentary hypersecretion may occur even after an exclusive diet of meat and soup. The cases described by Strauss seem identical with those reported under the designation of "Larval Hyperacidity," in which the acidity is at its height a half-hour after the ingestion of the test breakfast, diminishing from this time on as the thinning osmotic fluid is poured out, the resulting test breakfast being abundant and watery in the proportion of four or more parts of liquid to one of sediment.

It has been attempted to differentiate between these cases of larval hyperacidity and alimentary hypersecretion by the supposition that in larval hyperacidity the cause is to be found in a deficient starch digestion, producing excessive flow of gastric juice and thinning fluid which does not take place after an exclusive diet of meat, while in alimentary hypersecretion a deficient starch digestion is not essential, so that the excess of gastric juice represents a pure secretion unmixed

with thinning fluid. In the former condition the total acidity of the test breakfast is often normal or below normal, whereas in the latter form an increased acidity is encountered. This attempt to differentiate between these two forms of secretory excess is interesting, but is of no practical value at the present time. There is no doubt, however, in the writer's mind that when we know more about the thinning fluid and the conditions under which it is poured out, many obscure cases of alimentary hypersecretion will be explained.

It is difficult to explain such a case as the following without regarding the occurrence of an excessive flow of thinning fluid as probable.

E. C., aged twenty years, well-built athletic girl, came for diagnosis because of acne of the face and an undue sense of fatigue after riding horseback. There were neither nervous, gastric, nor intestinal symptoms. and aside from a moderate anemia no physical evidence of disease could be demonstrated. The fasting stomach was regularly empty; test breakfast constantly showed a quantity ranging from 240 to 230 c.c. of a watery consistency, separating on standing into two layers, the supernatant fluid being from three to four times the depth of the sediment. The total acidity was 10, free hydrochloric acid not present.

Many of the writers agree that although mild motor errors such as atony may be present, obstructive motor errors are accompanied not by alimentary hypersecretion but by the chronic form in which the fasting stomach contains acid fluid. Nevertheless, in two of Boas' cases it was possible for him to note the transition of a chronic hypersecretion into a pure alimentary form.

Von Huppert has suggested gastric ulcer as a cause for the ailment, while Elsner states that in his experience such an occurrence has never been noted. In the author's experience ulcer has apparently been a very frequent cause for alimentary hypersecretion. Owing to the comparative rarity of the ailment and the obscurity which envelops it, it may not be amiss to give the following examples of cases apparently due to these causes.

G. McA., aged fifty-five years, was well until twelve years ago, when after several months of distress in his stomach occurring several hours after meals, he vomited a large quantity of blood. He was successfully treated for ulcer and remained well until two years ago, when he again had distress two hours after eating, lasting until he ate again. He was put on the von Leube ulcer cure and recovered. For the past year his only complaint has been a slight feeling of distress, "as if he had gas on the stomach" whenever he is constipated. Physical examination showed no abnormalities. Fasting stomach empty. Test breakfast 210 c.c., the upper liquid layer constituting nineteen-twentieths of the entire meal.

W. P. L., male, aged twenty-six years. Two years before admission developed pain in the epigastrium, vomiting, and hematemesis. Was in the hospital for six weeks on an ulcer cure, and then remained well for one year. A year ago he developed heart-burn three or four hours after eating, relieved by eating, for which he desired treatment. The physical examination was negative. Fasting stomach empty. Test breakfast 190 to 240 c.c., the upper liquid layer constituting nine-tenths of the entire bulk. Total acidity ranged from 100 or 116, free hydrochloric acid 90 to 96. Three months later he had a large hematemesis with tarry stools and was again treated as ulcer. Since then—a period of six years—he has had no subjective symptoms, but his alimentary hypersecretion and high acidity have continued unchanged.

A. S., aged twenty-eight years. Two years ago developed pain of a burning character one or two hours after meals, lasting until he ate again. Occult blood present in the stools. Was treated medically for duodenal ulcer and remained well until one month ago, when the old trouble returned. Physical examination negative. Fasting stomach empty. Test breakfast 150 to 200 c.c., the liquid layer constituting nineteen-twentieths of the bulk. Total acidity 94 to 120, free hydrochloric acid 76 to 94. Patient was put on a von Leube ulcer cure and the symptoms ceased, and for the past eight years have not returned. The alimentary hypersecretion, however, continues as before.

Frequency.—The disease is comparatively infrequent. Boas speaks of 12 cases as comprising his series. Strauss¹ states that he has seen over 100 examples of the complaint. The writer's series embraces but 18 indisputable examples of the disease.

Symptoms.—Almost the entire literature of alimentary hypersecretion comes to us from Berlin, by Elsner, Strauss, Boas, Zweig, and others. The description which these writers give of alimentary hypersecretion, as they have seen it, is so different from that observed by the author that it seems best to give their views first and then to supplement their description by that of the cases which he himself has observed.

The principal symptoms observed by Boas and others are:

1. A remarkable loss of weight.
2. A great variety of neurotic symptoms, both general and local.
3. Obstinate constipation.

Emaciation is almost universal, a loss from 10 to 50 kilograms not being at all unusual, being only equalled by the emaciation of cases with advanced motor errors and food stagnation. Boas attributes the loss of weight to the loss of fluid, which he estimates at about 2 liters during the twenty-four hours. Strauss cannot explain the reduction

¹ Deutsch. med. Woch., April 11, 1907.

in weight by a loss of fluid from the system, as owing to the infrequency of vomiting the fluid passes into the intestine and is there reabsorbed. On the other hand, he attributes emaciation to the nervous dread of distress following eating, which he has noticed in many of his cases and which he terms "*citophobia dolorosa*." He rejects the possibility of the lack of proper nutrition being caused by diminished starch digestion by assuming that the intestine is capable of performing compensatory work. Nervous phenomena seem almost always present and are of a varied character, comprising pressure, fulness, eructations, excessive flow of saliva, violent pains, and occasional heart-burn. Nausea and vomiting occur exceptionally.

Constipation is often so extreme that response is difficult either by enemas or by the most violent and active medication. It is interesting that according to the German reports excessive phosphaturia, such as occurs with chronic hypersecretion, has not been noted.

The writer's experience in his cases has not been that of the Berlin school. In none of his cases was emaciation marked, nor were nervous phenomena, either general or local, in evidence. According to the clinical history the patients could be divided into two groups.

1. The history resembled that of gastric or duodenal ulcer, heart-burn, or distress appearing two or three hours after eating at the height of the digestion, the length of time after eating depending upon the quantity and the quality of the food. This distress continues until the stomach becomes empty or until the patient ate or takes soda. In these instances a differential diagnosis from ulcer by the history alone was impossible. Vomiting was but rarely observed and nausea seldom occurred. The bowels were moderately constipated but could be readily controlled by medication.

The intensity of the symptoms is proportionate to the height of the acidity. Alimentary hypersecretion with diminished acidity has not in the writer's experience given rise to any form of gastric distress.

Patients of this group that were treated as ulcer regularly lost their symptoms so that the disease passed into a stage that could be considered latent were it not for the characteristic appearance of the test breakfast.

The disease in the second group of cases ran either a latent course, so that the ailment was discovered only by a routine examination of the gastric contents, or the complaints apparently dependent upon the oversecretion were slight and insignificant. Occasional heart-burn and a moderate sense of fulness or distention were the most frequent symptoms noticed.

In this comparatively latent group are included those in which the more active symptoms described in the first set of cases subsided after

appropriate treatment. Some of the cases so treated have been followed during six or eight years by perfect gastric comfort, and during this time the patients have made no complaint of their digestions and considered themselves perfectly well, although the test breakfasts still show the characteristics of alimentary hypersecretion as marked as before.

Diagnosis.—Physical Signs.—There are no physical signs by which the disorder may be recognized. Frequent association of alimentary hypersecretion with gastropotosis and the enteroptotic habitus quoted by the German authors was not noted in the experience of the writer. Succussion sounds may or may not be readily elicited but possess no diagnostic value.

Gastric Analysis.—Fasting Stomach.—The fasting stomach is empty or practically so, and this point alone sharply differentiates between alimentary hypersecretion and the continuous form which depends on motor error.

FIG. 116



Test breakfast in alimentary hypersecretion. Layer of liquid three times the depth of the sediment. Breadstuffs thoroughly digested. Moderate amount of pharyngeal mucus. The fasting stomach in this case was empty. Measurements in centimeters.

Test Breakfast.—The appearance of the test breakfast is quite characteristic of the disorder. The bulk of the gastric quantity is excessive,

ranging frequently from 200 to 400 c.c. and is composed in large part of liquid. On standing the sedimentary layer is seen to be in depth equal to that observed in normal cases, but the supernatant layer is from 3 to 20 times that of the layer of solids.

It is fair to assume that alimentary hypersecretion may be said to exist whenever the fluid layer is 3 times the depth of that of the sediment, provided that the fasting stomach be empty. The total acidity varies. The majority of the cases show a marked increase in total acidity, often from 90 to 120, due to the presence of hydrochloric acid in the free and combined form. Proteid digestion in these cases is good, even excessive. Starch digestion on the other hand is impaired so that the maltose reaction is absent, while the erythrodextrin and especially the amidulin reaction is marked. In other cases the total acidity may be normal or subnormal. In one of the writer's cases the total acidity was but 10. These are regarded by the writer as examples of excessive secretion of the "thinning fluid" incompletely or completely neutralizing the hydrochloric acid. When the acidity is thus reduced, starch reactions are normal.

In order to demonstrate that the excessive fluid represents a secretion and not in part a retention of the liquid given with the ordinary test breakfast, Boas has recommended a dry test breakfast without any water being given. As the ordinary breakfast roll contains 35.5 per cent. of water he uses Albert biscuits, which contain but 9.8 per cent. of water. If five Albert biscuits are given without water under normal conditions the test breakfast is scanty and of gruel-like consistency. In alimentary hypersecretion from 100 to 200 c.c. of gastric contents are obtained, showing on standing a liquid layer three or four times the depth of the sediment.

Treatment.—In patients who present ulcer-like symptoms, medical treatment is of service, often totally relieving all sources for complaint. In mild cases the treatment consists of lavage with silver nitrate solutions, alkalies, and diet. Lavage with 1 to 3000 solution of nitrate of silver may be employed every second day and continued as long as apparent improvement results. The most convenient time for the lavage is late in the forenoon or in the afternoon before dinner at a time when residual food is scanty in amount compared with the excess of gastric juice. The solution may be gradually increased to 1 to 1000, but should be discontinued temporarily if diarrhea occurs. Alkalies are of service in reducing excessive acidity and in relieving the consequent distress. Owing to the excessive liquid bulk of the gastric contents alkaline powders are preferable to alkalized mineral waters. A "cure" at alkaline mineral springs is contraindicated. Boas recommends sodium citrate in dram doses 3 times a day at the height of digestion.

The diet depends largely upon the question of acidity. In high acidity the starches should be reduced and the fats and sugars correspondingly increased. Meats are generally well digested, but it is preferable to prohibit the red meats and to rely on chicken, fowl, ham, and fish. Bouillon and soups containing meat stock should be interdicted. There is some dispute as to whether it is advisable to allow three meals a day of average bulk, or more frequent meals in small quantities. The writer's personal preference leans decidedly toward frequent small meals, so that the acid generated by the one meal is combined with or neutralized by the succeeding meal taken two or three hours after.

The writer has employed belladonna and atropine hoping to inhibit oversecretion, but has observed no beneficial results from their use.

Unless the symptoms rapidly improve within a few weeks, it is advisable to place the patient on the regular von Leube ulcer cure, giving alkaline powders whenever it is necessary to relieve distress.

Under one or the other form of treatment above mentioned an improvement may confidently be expected in the subjected symptoms. The patient may feel perfectly well and free from all gastric distress and may continue in this state of well-being for years, although examinations show that the alimentary hypersecretion continues to the same extent. In some of these convalescent patients the symptoms may return with or without apparent cause and then the former treatment must be resumed until the ailment becomes again symptomless. The writer has no knowledge of any patient cured of this ailment by surgical means.

CHAPTER XX

NEUROSES

IN the older treatises on diseases of the stomach many more pages were given up to the description of nervous affections of the stomach than in books of a similar character more recently published. As medical diagnosis has perfected itself, largely through the experience gained by surgeons in abdominal operations, various forms of indigestion formerly classed among the neuroses are now shown to be dependent upon demonstrable organic disease. It is only in the last few years that chronic appendicitis even without physical signs or the classical history of definite attacks, has been demonstrated to be the cause of a large number of gastric complaints which up to this time were unexplained and for that reason considered to be of nervous origin. Lesions of the gall-bladder have also been placed upon a more definite basis and have been shown to be the exciting cause for reflex disturbances of the stomach hitherto obscure. Painful sensations formerly described as gastralgia or neuralgia of the stomach are now referred to gastric or duodenal ulcer. Further instances of the change in our attitude toward nervous affections of the stomach might be given, but the above examples are sufficient to show how accumulating experience has resulted in transferring many forms of indigestion from the neurotic group to that of definite organic disease.

The frequency of nervous indigestion is impossible to determine with any accuracy owing to the extreme difficulty of ruling out organic affections. This difficulty is often intensified by the predominance of nervous symptoms in susceptible patients who are ill from organic disease. Sufficient time for observation, a careful study of the case, the temperamental susceptibilities of the patient and his reaction to environment and oftentimes repeated physical examination, may be necessary before the diagnosis is reached, and in the meantime the symptoms are regarded tentatively as of nervous origin. Many patients pass so soon from observation that sufficient opportunity is not afforded for diagnosis and they are entered on the case books as instances of nervous indigestion, simply because that was the first working diagnosis. A large number of so-called nervous indigestions occur with visceral ptoses. While there is no doubt of the existence of neurasthenic dyspepsia in these patients, they should not be regarded as suffering from nervous indigestion alone.

The older estimates were that 50 to 75 per cent. of all patients suffering from indigestion were examples of the nervous form. These figures seem to the writer altogether too high, even among those classes in the community such as Polish Jews who are peculiarly and racially susceptible to neurasthenic disturbances.

The majority of the available statistics were compiled before the era of modern surgery and its revelations of organic disease formerly unsuspected. The writer estimates that in his cases approximately 15 per cent. of dyspepsias may be regarded as of purely nervous origin, closely approaching the figures given by Fenwick, who found nervous indigestion in 3 per cent. of his hospital cases and in 13.2 per cent. of his private series. An additional 10 per cent. should be added if there be included patients with the enteroptotic habitus and well-developed visceral ptoses.

Diagnosis.—Although the group of nervous diseases of the stomach is a diminishing one, it is nevertheless one of the most common sins of diagnosis to declare that patients are suffering from nervous indigestion when they are really the victims of organic disease. In some cases this error in diagnosis is justifiable, as the mimicry of disease may be so perfect that without an exploratory operation a definite diagnosis between organic and functional disorders cannot be made. In the great majority of instances, however, the error is a culpable one, due to lack of thoroughness in the examination of the patient, and could have been averted had attention been paid to the following rules for the examination of the case.

Too much reliance should not be placed upon the patient's own statement of his complaints without a cross-examination that might bring out the salient symptoms of organic or associated disease. The previous history is frequently as important as the narration of recent events.

It is a sad commentary on our diagnostic methods that so many dyspeptics are treated without there being made at any time a physical examination, even of the abdomen. Although a careful physical examination of the abdomen is essential, it is almost equally important to make an examination of the heart, lungs, and arteries, to examine blood pressure, the urine, and the peripheral reflexes. The examination, therefore, should be thorough, complete, and repeated.

There seems to be a general neglect of the importance of gastric analysis, or a reluctance on the part of the physician to force such an examination upon his patient. The writer's experience is that patients are more than ready to have a test breakfast examination made if the reason for such a procedure is explained to them.

Before the diagnosis of nervous indigestion is made organic disease

must be ruled out by a carefully taken history, by thorough and complete physical examination and in the majority of cases by examinations both of the fasting stomach and of the test breakfast. To simplify this exclusion of organic cause for the patient's complaint the writer would suggest the following rules:

1. A diagnosis of nervous indigestion should not be made in any case in which the fasting stomach contains more than 30 c.c. of fluid giving a reaction for free hydrochloric acid. Hypersecretion is a rudimentary form of pyloric stenosis, spasmodic or organic, and is regularly due to an organic and demonstrable cause.

2. The diagnosis of nervous indigestion should not be made in case of persistent hyperacidity accompanied by epigastric pain. Nervous hyperchlorhydria may occur, but is not accompanied by either heart-burn or pain. The association of either of these latter symptoms should suggest an organic origin for the complaint.

3. Achylia may be of nervous origin but is not then accompanied by serious motor error. Achylia with food stagnation indicates in all probability cancer of the stomach.

4. Achylia accompanied by pain or vomiting indicates an organic cause for the secretory disorder, as these symptoms do not occur in the functional cases.

5. The diagnosis of nervous indigestion should never be made when gross recognizable food remains are repeatedly found in the fasting stomach. Under the influence of mental shock, nervous dread, excessive fatigue, the motor functions of the stomach may be temporarily abolished, so that food remains within it for an abnormal period of time, but such a loss of motility is temporary and cannot be demonstrated in succeeding examinations.

6. The diagnosis of nervous indigestion should never be made when epigastric pain recurs at a definite time after eating. The old-fashioned diagnosis of gastralgia or neuralgia of the stomach will almost invariably be found wrong.

7. The diagnosis of nervous indigestion should not be made when one symptom alone persists without other evidences of nervous instability. Nervous disorders of digestion are multiform and varied, irregular in character, and usually associated with other general neurotic manifestations. It is improbable that in a patient of good nerve poise and self-control, one gastric symptom, be it nausea, flatulence, pain, or distress, should persist day after day without other local symptoms, and without other concomitant evidences of a nervous disorder. Such a clinical course regularly implies organic disease.

8. Symptoms that might indicate a nervous disorder may be the result of drug addiction, and in doubtful cases drug habits should if

possible be excluded before the complaint can be said to be of purely nervous origin.

9. The diagnosis of nervous indigestion should not be made when complaint is made of indigestion by those over forty years of age in whom the disorder appears for the first time. Those who have passed through the greater part of the storm and stress period of life without having shown their inability to withstand the effects of their environment, are pretty well "seasoned" as they approach middle adult life, and are examples of the nervous survival of the fittest. The diagnosis of nervous indigestion under these circumstances will almost invariably be found to be wrong.

10. Although the existence of an organic cause for the ailment may be demonstrated, it does not necessarily follow that all the symptoms in the case are to be explained on this basis. Nervous people may be afflicted by an organic disease, and those with organic disease may be thereby rendered weak and nervous, so that in many cases organic and nervous symptoms are intermixed to form a complete clinical picture that is often quite confusing.

The greatest assistance in unravelling the problem is rendered by the presence of the stigmas of the enteroptotic habitus in the patient. Those with broad costal angle and robust and well-knit frame rarely suffer from prolonged functional ailments. Those on the other hand of delicate build, with narrow costal angle and visceral ptoses are inherently the victims of a universal congenital neurasthenia and are almost regularly subject to functional disturbances of digestion whenever they run down. Should organic disease attack such a patient a certain proportion of the symptoms at least are apt to be of an associated functional nature.

Symptoms.—Nervous affections of the stomach have certain characteristics in common which may be of service in elucidating the diagnosis.

1. The symptoms are shifting and variable. Instead of one symptom steadily predominating as in organic disease the clinical picture changes almost from day to day. There may be nausea today, fulness and distention tomorrow, and then a comfortable digestion for several days succeeding. The variety in the clinical symptoms is suggestive of nervous disorder.

2. The intensity of the symptoms may bear little relation to the character of the food. The most indigestible assortment may be eaten with relish and without discomfort while at other times the simplest food may provoke considerable distress. The lack of regular association of distress with the time, character, and bulk of the meal is suggestive of gastric neurosis.

3. The symptoms of nervous indigestion are often dependent upon the nervous state of the patient, appearing during overexcitement or fatigue or accompanying periods of worry and apprehension, and disappearing when the patient is again happy and contented. In some instances symptoms only appear before or during the menstrual period, even though that function be normal in other respects. The dependence of the symptoms upon the nervous state of the patient must not, however, be regarded as convincing proof for the functional origin of the ailment, as in many organic diseases of the stomach the nervous system bears an important share in influencing the severity of the complaint. As an extreme example may be mentioned instances of cancer in which symptoms, aggressive and severe, have disappeared entirely under the influence of nervous or physical shock, or of mental exaltation, such as that accompanying religious revivals. It is almost unnecessary to allude to the fact that the symptoms of nervous patients who are ill from any organic disease may be greatly influenced by an underlying neurosis.

4. Symptoms that readily yield to an antineurotic treatment are presumably of nervous origin.

Therapeutic Tests.—Three therapeutic tests may be mentioned.

(a) Symptoms that readily and completely yield to small doses of bromide are probably on a nervous basis. The following prescription has been found invaluable for the purpose.

R—Chloral hydrat.	℥ss
Strontii bromid.	℥iiss
Aq. chloroform.	℥iv
Spirits anisi	gtt. viij
M. Sig.—Teaspoonful in water four times a day.	

A week's trial of this prescription is of great service in cutting out from a given case those symptoms that are of a purely neurotic character.

(b) A large proportion of patients with nervous indigestion are suffering from chronic starvation, due to insufficient food. They say that they cannot eat more than they do because of distress. If in such a case the physician be able to gain the patient's confidence so that a more liberal diet can be given, and if it can be shown that sufficient and wholesome nourishment produces no greater distress than the invalid foods to which the patient has been accustomed, the evidence is suggestive that the ailment is of a functional character.

(c) If gastric symptoms occur in a patient who is nervously run down, and disappear by rest and change of air and scene, to reappear later when the activities of daily life are reassumed, a functional origin for the complaint may be strongly suspected.

5. Symptoms of a generalized character are of importance in determining the diagnosis. With melancholia, hysteria, epilepsy, and with psychoneuroses of various kinds, concomitant gastric symptoms, in the absence of demonstrable organic disease, may be considered tentatively to be of functional origin. Sexual errors, such as interrupted coitus, masturbation, and excesses of all kinds, are often followed by symptoms of functional derangement of the stomach.

6. It must again be emphasized, even at the risk of what may be considered reiteration, that those who inherit the enteroptotic habitus are by nature predisposed to nervous indigestion throughout their lives whenever they overtax their nervous or physical strength, and the existence of the enteroptotic habitus is a strong presumptive proof that the symptoms presented are in part or in whole of a nervous or functional nature.

The symptoms of nervous dyspepsia may be divided into three groups, according to whether the functional error concerns sensation, motility, or secretion. It is, however, often impossible to draw a definite line between these three groups, as in a given case the symptoms of each group may be present so intermingled as to form a confused clinical picture. Nervous indigestion after all is but the local manifestation of a general nervous disorder, and the symptom-complex is not complete without the presence of neurasthenic or psychoneurotic symptoms that may or may not overshadow the local evidences of indigestion. The individual symptoms of nervous indigestion may now be described.

SENSORY NEUROSES

Disorders of the Appetite.—Bulimia.—Bulimia, occasionally termed cynorexia, consists of an impulsive and spasmodic sensation of hunger that cannot be resisted. The patient will suddenly feel an uncontrollable craving for food and will grow pale or may even faint unless food is at once obtained. The craving is so irresistible that the patient may forget all conventional ideas of decency and will eat anything that first comes to hand and in any place. One patient of the writer's when seized by this impulse would run to the nearest ash barrel and eat anything that he could pick out.

The attacks pass usually with the first mouthfuls taken, so that, as a rule, an excessive quantity of food is not required. There is, therefore, a difference between polyphagia or gluttony and bulimia, although the two may be combined.

Whether the hunger centre is stimulated centrally or peripherally is clinically quite unimportant. The disorder usually occurs as a

primary neurosis in hysterical or epileptic patients or in those who are the victims of a profound psychoneurosis. In rare instances bulimia has occurred as an early symptom of brain tumor.

Acoria.—Acoria consists in the absence of normal sense of satiety after eating, so that the patient never knows when hunger is appeased. Acoria may be combined with anorexia, so that the patient may eat without appetite but will keep on eating without any sensation that he has eaten enough. Acoria differs from bulimia in that there is no morbid eagerness for eating as in the latter condition.

Anorexia.—Anorexia is a common symptom in many organic affections of the alimentary tract, in many general diseases, such as tuberculosis and nephritis, and is a common concomitant of nervous states. As a symptom it has very little diagnostic value except in those of adult years who without apparent cause show an increasing repugnance to food. In these cases cancer of the stomach should always be suspected.

Nervous anorexia may appear as the result of acute grief or nervous strain. The patient is so overwhelmed by his trouble that he cannot eat. This is an experience common to all. In other instances the symptom is more permanent. The patient will sit down to his meals with reluctance, will pick at the food, and will be able to swallow a few mouthfuls with difficulty. After that he can eat no more. It is surprising, however, how many nervous patients retain their flesh and strength, though they apparently eat nothing. This form of anorexia must not be confused with the sensation of early satiety, which is a symptom of gastric atony. In this latter condition the appetite is usually good at the beginning of the meal but is easily appeased.

In the extreme cases of the affection, almost exclusively confined to the hysterical and insane, the repugnance for food may be so great that the patient cannot bring himself to eat any food at all. Loss of weight may be extreme and the patient may become a living skeleton. Recovery may take place after weeks or even months of inanition or the patient may die of starvation unless nourishment be sustained by forcible feeding through the tube.

Gastralgokenosis.—Gastralgokenosis (from *γαστράλγεια*, gastric pain, and *κενός*, empty) is the term suggested by Boas to describe the sense of painful emptiness of the stomach that occurs as a pure neurosis. Epigastric pain occurs whenever the stomach is empty is relieved at once by food. According to Boas the symptoms may be permanent or periodic. The writer has had no experience in cases of this type of a purely neurotic character. In various neuroses there may be vague sinkings in the stomach at any time of the day, occasionally but not regularly relieved by eating. In many of the writer's cases of chronic catarrhal gastritis with normal acidity, a sinking faintish feeling in the

epigastrium relieved by eating, was observed, but this symptom was one of vague discomfort and not of pain. It seems to the writer that the cases described by Boas are probably instances of hunger pain due to gastric or duodenal ulcer or to hypersecretion from whatever cause it may be induced. It has occurred in a number of the writer's series of chronic appendicitis, disappearing after operation.

Hyperesthesia.—Hyperesthesia of the stomach may be defined as a condition of hypersensitiveness to normal gastric contents, so that symptoms of distress will accompany digestion, although the stomach be free of organic disease and the secretions may be normal. In this condition the simplest food may cause distress.

The most common symptoms are fullness and a sense of distention after eating, often amounting to actual pain. There may be burning sensations in the stomach, which we ordinarily call heart-burn, but which may not be accompanied by any increase over the normal acidity. Indeed, in many instances in which this complaint is made the gastric contents may be subnormal or even devoid of all acid whatever. Paresthesia may occur, such as a feeling as if the stomach were being scratched or that it crackled as though it were stiffly varnished. Nausea is a common enough symptom and vomiting frequently occurs, more often, however, induced than spontaneous, relieving the distress. There may be the feeling of gas in the stomach, which the patient attempts to dislodge in every conceivable way. The symptoms occur in attacks extending over several days or weeks, often precipitated by periods of nervous excitement or fatigue. An unfortunate result of the ailment is that the patient regards his distress as due to excessive eating or to food which ferments or turns to acid, and resorts to a diet that is quite insufficient.

The ailment is said to be quite common among nervously inclined individuals, in those addicted to the abuse of tea, coffee, and tobacco, or the victim of drugs, especially of opium and cocaine. Nicotine poisoning has been said to be an exciting cause for the ailment. It may also occur as a temporary phenomenon after excess in eating and drinking.

Visceral sensibility is regularly intensified in hypochondriasis and in irritable conditions of the central nervous system that accompany neurasthenia and anemia. Many cases of hyperesthesia of the stomach are difficult to explain, as it has been proved that the alimentary tract from the commencement of the esophagus to the junction of the rectum with the anal canal is completely insensitive to tactile stimulation and that the mucous membrane of the esophagus and stomach in health and in disease is totally insensitive to the contact of acids. There is, however, a sensibility of the lower end of the esophagus to heat

and the feeling produced by swallowing hot fluids may be referred entirely to the epigastrium. The stomach itself is not sensitive to either heat or cold. The lower end of the esophagus is extremely sensitive to alcohol, although the introduction of concentrated solutions of this substance into the stomach produces merely a sensation of warmth. Pain in the stomach may apparently be due to hyperacidity, not as was formerly supposed by its burning effect upon the gastric mucosa, but by reason of the increased peristalsis which it provokes, raising the intragastric pressure and producing tension upon the stomach wall. To cause pain this tension must be rapidly produced, otherwise there is merely a sense of fullness. It is conceivable that in susceptible patients the motor activity may be so increased that painful or uncomfortable sensations may be produced by dilutions of hydrochloric acid of a less strength than is normally present in the gastric juice. It is a matter of clinical observation that very weak solutions of hydrochloric acid will produce discomfort or pain in certain people so that an individual intolerance for acids has been suggested as one of the causes for nervous dyspepsia.

Clinical Types.—The following clinical types of gastric hyperesthesia may be enumerated:

Fulness or gastric pain may follow meals that are rapidly eaten. The stomach is normally in a state of partial contraction, or rather of unrelaxation, which we call "tone." To accommodate the bulk of a meal there occurs a relaxation of the circular muscle fibers followed by a gradual rearrangement of these fibers, so that instead of the stomach wall consisting of from fifteen to twenty layers in the empty state, there are but two or three layers when the organ is full. In rapid eating intragastric pressure rises before there is sufficient time for the muscular fibers to relax and so to rearrange themselves as to allow sufficient room for the food. In atony the muscular fibers are more or less completely relaxed before the meal is taken, so that any increased dilatation can occur only through rearrangement of the muscular fibers, and as this can result only from increase in intragastric pressure, distress occurs.

It is, therefore, evident that the more rapidly a meal is eaten the greater is the tendency toward fulness and discomfort. A small meal hastily bolted will give greater distress than a large meal taken slowly. Whenever relaxation of the muscle fibers is impaired, whether from ulcer or other forms of infiltration of the stomach wall, by adhesions, or by reflex inhibition of relaxation that may occur with chronic appendicitis or diseases of the gall-bladder, a sense of fullness and discomfort may attend the taking of meals, even of small size. It is interesting to note that the sensation of fullness may result from the two opposite conditions—hypertonus and atony.

When air is swallowed slowly and naturally, the gradual dilatation of the stomach accommodates the larger air bubbles without discomfort, but when gas in the stomach increases rapidly with hasty eating a corresponding relaxation of the muscle fibers cannot occur soon enough to obviate considerable distress. A rapidly eaten and imperfectly masticated meal regularly causes an increased peristalsis with inhibition of pyloric relaxation, caused by the presence of hard masses of food. The distress will continue until the undigested particles are softened and even then an increase in peristalsis may be required before they can be forced through the pylorus. Relief naturally is experienced when the stomach is emptied by lavage or by vomiting. The writer has gone thus deeply into the affects of rapid eating and insufficient mastication because in his experience the majority of patients with so-called gastric hyperesthesia suffer from the mechanical effects of their mode of eating. It is hardly fair to describe these errors as nervous indigestion.

There are patients who are peculiarly sensitive to hot fluids and certain kinds of food and drink. Soup, tea, and coffee must be cooled before they can be taken without discomfort, and if taken too hot there results a burning feeling running down the esophagus to the stomach instantly relieved by sipping cold water. Fluids, syrups, and acid vegetables, such as tomatoes, will produce the same result. Alcohol will almost regularly precipitate an attack, especially the more concentrated beverages, such as sherry, brandy, Madeira, and the cordials.

The symptoms of this group seem to be produced by irritation of the lower portion of the esophagus rather than by irritation within the stomach itself. This view is supported by Hertz's experiments showing that painful sensibility of the lower end of the esophagus may occur by thermal and alcoholic irritation and by the fact that even teaspoonful doses of cold water will immediately alleviate the distress.

This form of hyperesthesia in the writer's experience is regularly due to one of two causes and can therefore be hardly considered a true neurosis.

In the majority of instances the symptom-complex accompanies gallstones or irritative lesions of the gall-bladder. The writer has seen a number of cases in which the symptom has disappeared after the passage of gallstones or after the gall-bladder has been drained. The following history will illustrate this point:

G. H., aged forty-seven years, was practically free from indigestion until he had typhoid fever twelve years ago. Since that time he has complained of recurring attacks of pain and tenderness over the gall-bladder with nausea and vomiting, each attack lasting about twenty-four hours and then reappearing after an interval of several weeks.

Between the attacks there has been at times a dull aching pain of the liver running to the back. During these twelve years the patient has been unable at any time to take soup, tea, or coffee unless lukewarm. Liquids hotter than this as well as the more concentrated forms of alcohol will produce an immediate burning pain running from the throat to the stomach instantly relieved by hot water, so that at his meals he has to sip cold water frequently to relieve his distress. This symptom continued until the gall-bladder was drained and 8 ounces of mucopurulent fluid and a number of gallstones were evacuated. Since then he has been totally free from his complaint.

In other instances gastric or esophageal hyperesthesia is dependent upon gout, and the symptoms will continue until relieved by appropriate treatment. It is suggestive in these cases that the throat is the most sensitive and shows the characteristic appearance of gouty pharyngitis.

Gastralgia.—"Gastralgia" or pain in the stomach and "epigastralgia" or pain referred to the epigastrium are terms used to describe painful sensations in the upper part of the abdomen. The term "gastralgia" is an unfortunate one, as it implies that pain arises in the stomach itself, but it has been sanctioned by usage so as to indicate merely that the pain is referred to the neighborhood or region of the stomach. It is in this broader sense that the writer employs the term.

Gastralgia was formerly regarded as a frequent complaint occurring in periodical attacks quite independent of any organic disease. Attacks have been described of great severity. The patient will be suddenly seized with a burning, boring, tearing, or lacerating pain, originating in the epigastrium and radiating in various directions. The face is anxious and drawn, the pulse rapid and thready, the temperature subnormal. Faintness may occur or the patient pass into a mild form of collapse. The seizure, however, ceases somewhat abruptly and recovery is uneventful until another paroxysm occurs. Vomiting and repeated retching are not infrequent. The writer is extremely skeptical as to the occurrence of paroxysms of this severity that can be considered of a neurotic character. There is no doubt that epigastric pain may occur as a pure neurosis in nervous or hysterical patients, but the pain is rarely severe, is not limited to the epigastrium, but occurs in other parts of the body, showing a general neuralgic condition.

Occurrence.—Before we are warranted in assuming that a pain referred to the stomach is nervous we must rule out all organic diseases of the stomach, such as ulcer, adhesions, or pyloric stenosis that are regularly accompanied by paroxysms of pain, acute appendicitis, the gastric crisis of tabes, biliary colic, angina abdominalis, epigastric hernia, and inguinal hernia that is due to a patulous condition merely of the internal ring. The possibility of lead poisoning must also be

considered. Malaria may give rise to recurring epigastric pain often with vomiting, which usually marks the onset of the paroxysm. The pain is rarely limited to the epigastrium, usually diffusing over the abdomen. Cabot¹ states that in a single week of service in the Massachusetts General Hospital, three patients were sent in to be operated on for supposed appendicitis. All had malarial fever and all were promptly cured by quinine. In some cases of uremia, upper abdominal pain may be observed which may be entirely located in the epigastrium and which may precede uremic convulsions. The late Dr. J. H. Musser² reported a case of uremia with vomiting and abdominal pain. The autopsy showed the pain could not be accounted for by any abdominal condition and was evidently toxic. It is not improbable, however, that in the uremic cases erosions or ulceration may be the cause for the pain.

Epigastric pain appearing whenever the patient walks after eating, is characteristic of arteriosclerosis, although the identical phenomenon may also be observed with perigastric adhesions.

A constantly recurring epigastric pain without other manifestations of disease should never be considered of nervous origin but should regularly suggest a dependence upon an organic lesion. When all the above causes for pain have been excluded there remains but a small number of patients whose pain may be considered of neurotic origin.

Insane and Feeble-minded.—Epigastric pain is frequently observed among insane and feeble-minded patients and may closely simulate organic disease of the stomach. The assumption that the epigastralgia is dependent upon the psychosis would be unwarranted were the patient of normal mentality.

Chlorosis.—Chlorosis generally accompanied by constipation may be the cause for sudden or nagging epigastric pain, sometimes coming immediately after meals, sometimes later. The pain may not always remain in the epigastrium but may shift to the lower abdomen, chest, or back. Some of these patients show hyperacidity while others do not, so that the pain cannot be said to depend upon the degree of gastric acidity.

A course of iron and saline laxatives will result in a speedy and uneventful recovery. The writer has seen a number of these cases, but regards them far less common than ordinarily supposed.

Syphilitic Disease.—Gastralgia may occur in syphilitic patients due to depreciation of their general condition rather than to any secondary or tertiary lesion. These cases are commonly seen in hospital practice and are readily relieved by antisiphilitic treatment.

¹ Differential Diagnosis, p. 140.

² Amer. Med., March 26, 1910.

Hyperesthesia.—Epigastric pain may occur in those with apparently normal digestion as the result of the so-called hyperesthesia of the stomach. These cases are described on page 545.

Pneumatosis Ventriculi and Aërophagia.—Epigastric pain as a neurotic manifestation may occur with pneumatosis ventriculi and aërophagia, and is elsewhere described.

Pain in the epigastrium is a common symptom in heart disease during the period of decompensation and is due to congestive swelling of the liver. Anorexia and vomiting are frequently concomitant symptoms. The frequency of this form of pain in hospital cases is shown in the accompanying table from Cabot.¹

CAUSES OF EPIGASTRIC PAIN

Gastric and hepatic congestion due to cirrhosis or cardiac disease	898 cases
Appendicitis	350 cases
Peptic ulcer	347 cases
Gallstones	329 cases
Hyperchlorhydria	326 cases
(Many of these cases may actually be cases of peptic ulcer. Only operation or autopsy can decide.)	
Gastric cancer	133 cases
Pericarditis	88 cases
Gastric neurosis	72 cases
Pancreatitis	7 cases
Pyloric adhesions	2 cases
Angina abdominalis	1 case

Violent abdominal pain may occur with diabetes, and may occur in the form of an abdominal crisis preceding coma. Downes and O'Brien² reported two cases of diabetes in which an abdominal crisis occurred with pain, vomiting, and abdominal rigidity, suggesting to two or three observers the necessity for operation.

Severe attacks of epigastric pain may occur in those addicted to morphine or cocaine, and these drug addictions should regularly be suspected in doubtful cases of apparent neurotic origin.

Flatulence.—A certain amount of air is always present in the healthy stomach contained within that portion of the fundus which lies above the cardiac orifice, forming an air chamber known to the radiologists as the "magenblase." Chemical examinations shows the contents of the air-bubble to be composed of ordinary atmospheric air which has been swallowed with each muscular effort at deglutition either during the meals or between meals with the saliva.

¹ Differential Diagnosis, 1911.

² Intercolonial Medical Journal, Australasia, September, 1909.

There is undoubtedly possessed by the stomach a certain aspirating power so that air is sucked into the organ by the alternate contraction and relaxation of its wall, but the amount of air thus introduced is small compared with that entering with deglutition.

Etiology and Symptoms.—Ordinarily no symptoms are produced by the average amount of air contained in the “magenblase,” although occasionally hypersensitive or imaginary individuals claim that they are distressed by flatulence and constantly attempt in every conceivable way to relieve themselves, although physical examination or radiographs may show the amount of gas to be practically negligible.

Increased amounts of air may be introduced by too frequent attempts of deglutition, such as with salivation, nervous habits of aimless swallowing, irritable conditions of the throat and acid feelings in the stomach. The chewing of tobacco and hasty eating of unmasticated food are frequent causes for excessive accumulations of gas.

When air is gradually introduced into the stomach, relaxation and rearrangement of the muscular fibers may allow the organ to adapt itself to the increased volume of its contents, so that intragastric pressure is not unduly raised, and consequently no great distress is experienced. When, however, the gaseous volume is rapidly increased, fulness, distress, and even pain may be produced before the stomach is able sufficiently to dilate. Rapid eating is one of the commonest causes for gastric flatulency. Lack of adaptation of the stomach to its contents occurs with infiltration of the wall by inflammatory tissue or malignant growths, or with perigastric adhesions, so that with ulcer, cancer, and adhesions a moderate, even a normal quantity of gas may occasion severe distress.

Hypertonus, such as occurs with gallstones, cholecystitis, and chronic appendicitis, may likewise produce painful sensations of fulness and distention even though the actual amount of gas be not excessive.

Flatulence is favored by the opposite condition of hypertonus, and is a regular accompaniment of atony and of atonic gastroptosis, constituting the most constant symptom of these ailments. The atonic gastric wall allows the stomach to be easily inflated. The bulk of the gas naturally depends upon the pressure to which it is exposed, and as the reduction in pressure allows it to increase in bulk, the air-bubble or “magenblase” in atony is regularly larger than normal.

The amount of gas is regularly most voluminous in the atonies that are accompanied by hyperacidity, probably because of the instinctive desire to neutralize the acid by swallowing saliva and the consequent pumping in of air. The gaseous distention is furthermore increased by the carbon dioxide that is liberated whenever the patient takes soda. A relaxed atonic stomach cannot readily expel the gaseous contents and

hence there results an increasing accumulation. The occurrence of extreme distention accompanying parietic conditions of the stomach has been described in full under acute dilatation.

Whenever intragastric pressure is raised either by accumulation of gas, or by an increased pressure upon the contents, a sense of fulness or distention is experienced, which is attributed by the patient either to overeating or to "fermentation" of food. The natural tendency is for the patient to restrict himself to a diet that is often quite insufficient for the maintenance of health and nutrition and to limit his dietary to articles of food that are not readily fermentable. A popular misconception is that if the eructated gas bear the odor or taste of food that has been eaten, the proof is convincing that the food is not properly digested, and so one article after another is cut out from the menu. As long as any seasoned or flavored food remains in the stomach, just so long will the gas that is raised carry the flavor upward. It is only when abnormal odors are detected, such as hydrogen sulphide or the rancid odor of butyric fermentation, or when the ordinary flavors of ingested food are repeated long after the stomach should be empty, that the symptom demands investigation.

Flatulence is a common symptom of cardiac disorders during the stage of decompensation. Fulness and distention occur after meals, and the accumulation of gas interferes with the descent of the diaphragm and occasions dyspnea, palpitation and irregularity in the heart's action. Anorexia often to the point of food loathing is commonly present and vomiting due either to venous engorgement of the stomach or to injudicious medication may be repeated and difficult to control. Pain caused by enlargement of the liver is added to the symptom-complex so commonly observed in our hospital wards.

Flatulence frequently occurs with constipation and is relieved when the bowels are opened, but in these cases intestinal distention precedes the gastric distress.

Gaseous accumulations may be produced by either carbohydrate fermentation or proteid decomposition, but the gases are generated so slowly that sufficient time is allowed for the stomach to adapt itself to the increased bulk and but little distress ordinarily ensues. Fermentation, moreover, rarely produces a sufficient volume of gases to be a disturbing factor. The odor and taste of the eructations may be exceedingly unpleasant and may create a loathing for food, but the feeling of fulness or distention is but rarely experienced.

An appreciable fermentation occurs only with grave motor errors, usually due to pyloric stenosis, and owing to the inhibitory effect of hydrochloric acid upon fermentative processes, more common in the malignant cases.

The term "fermentation" is unfortunately applied to any or all forms of gastric flatulence, and patients are overdieted accordingly and restricted in their choice of food. The diagnosis of "fermentation" in the absence of grave motor error is almost invariably based upon misapprehension of facts and almost invariably wrong.

Nervous Flatulence.—Nervous flatulence may occur temporarily from many causes, such as mental shocks or emotional outbursts, but is rare as a more permanent phenomenon except in enteroptotic individuals with gastro-intestinal atony. The cause for the acute flatulence is probably a suddenly induced atony of nervous origin.

Sudden sharp attacks of flatulence may occur in nervous pregnant women who never vomit during the period of gestation. The ailment usually appears in short, sharp attacks ordinarily induced by fatigue, excitement, or mental shock. The attack is ushered in by oppression in the chest, and distention of the epigastrium which later becomes more generalized so that the entire abdomen is distended, tense, and tender. Eructations of odorless gas in large quantities and the expulsion of flatus are followed by temporary relief only, as the gas seems rapidly to accumulate. The attack may last for from several hours to one or two days, and often subsides somewhat abruptly, although considerable soreness of the abdomen may persist for several days. Subsequent paroxysms may recur at intervals of one or more weeks. In other cases the flatulence is more or less continuous and merely varies in degree from time to time, although the exacerbations are rarely sufficiently severe to occasion more than an average amount of distress.

Aërophagia.—Nervous eructations or aërophagia comprise a group of cases with well-marked clinical symptoms that are quite distinctive. The disorder consists in the involuntary swallowing of air that is immediately eructated with a loud and explosive noise. Gas-bubbles ascend the esophagus and burst in the pharynx with explosive force, one after another, often with almost incredible rapidity, occasionally every few seconds. Ordinarily, however, single eructations occur and are repeated at intervals of from five to fifteen minutes. The paroxysms cease during sleep, but persist during the meals, and are often brought on or intensified by anger, emotional outbreaks, or by visits from unsympathetic friends. An attack may often be precipitated by examination of the throat or by palpation of the epigastrium during the routine examination of the patient. The amount of gas brought up on each occasion is quite small—its constituency is that of atmospheric air.

The essential feature of the ailment consists of a clonic spasm of the pharynx which forces air involuntarily into the esophagus, and which is easily detected by placing the fingers upon the upper portions of the neck during a paroxysm. The greater bulk of the air does not usually

reach the stomach, but is contained in the esophagus before it ascends, although in many instances some air certainly does enter the stomach and is heard as a loud and distinct bruit or bursting sound following the pharyngeal spasm when the stethoscope is placed over the epigastrium. In rarer instances pharyngeal contractions may force air into the stomach in greater quantities than can be dislodged upward, so that the stomach becomes rapidly inflated. This condition is often known as *pneumatosis ventriculi*. Gas may even pass from the stomach into the intestines so that general abdominal tympany results.

In some cases nervous eructations occur in short but recurrent attacks, some of which are mild though annoying both to the patient and to those in the immediate neighborhood, while in others the eructations are excessive and the attacks so prolonged that the patient becomes quite worn out. The attacks may be separated by fairly long intervals, or the paroxysms may run together so that the disorder becomes more or less permanent, and exceedingly rebellious to treatment.

Vomiting.—Vomiting can only be considered a symptom of nervous dyspepsia when all forms of organic disease that may produce the symptom can be excluded from the diagnosis. Nervous vomiting must be distinguished from regurgitation of food which is ejected whenever it reaches the mouth. It is impossible to do more than briefly allude to a few of the more ordinary organic causes for vomiting which are to be considered as possibilities in any given case.

Etiology.—Organic diseases of the stomach are frequently accompanied by vomiting. Vomiting of acid watery fluid or of ancient food remains are readily referred to an organic cause. Erratic vomiting may usher in the symptoms of chronic ulcer or cancer, so that for a time the diagnosis may remain in considerable doubt. Persistent vomiting having many of the characteristics of the purely neurotic type may occur with chronic appendicitis. Irregular and often unpremeditated vomiting is not uncommon with gallstones even though they be otherwise latent.

Reflex vomiting may originate from various irritative lesions of the abdominal or pelvic viscera. In women uterine displacement, pelvic adhesions, and organic tumors are not infrequently the cause especially of vomiting that occurs before or during the menstrual period, although menstruation may be regular and painless. In men with enlarged prostates and retention of urine, nausea and vomiting frequently occur. Rosenberg and Herschell¹ alludes to these cases under the name of *Urokinetic Dyspepsia*, and consider them evidently of toxic origin.

¹ *Deutsch. Med. Woch.*, August 17, 24, 31, 1899; *Med. Press and Circular*, May 31, 1905.

Various toxemias are provocative of vomiting. Uremic vomiting seldom occurs, however, as an isolated single symptom, but is regularly accompanied by other symptoms and physical signs as well as by the urinary evidences of renal involvement. The vomiting may be accompanied by pain or diarrhea from uremic ulcers in the stomach or intestinal tract. In exceptional cases the vomiting of uremia may be persistent at the onset, obscuring the other symptoms of the kidney disease.

Pregnancy should always be excluded, especially difficult being the early exclusion of extra-uterine gestation. The vomiting may be that of the ordinary morning sickness, or may be of a pernicious type, with errors in the ammonia coefficient and slight jaundice that indicate parenchymatous degeneration of the liver. Prolonged vomiting may be perpetuated by starvation, acidosis, and acetonemia. In other cases, nervous pregnant women may vomit from time to time throughout the entire period of gestation simply because they are nervous, apprehensive, and afraid of what they will inevitably have to pass through.

Pulmonary phthisis may be accompanied by vomiting as an initial symptom. The vomiting usually occurs when the patient rises in the morning and is preceded by tickling in the throat and cough. Retching follows the effort to dislodge sticky tenacious mucus, and whatever there may be in the stomach will come up. In other cases every attempt to partake of food is followed by an attack of coughing which terminates in vomiting. The emesis is not, however, preceded or accompanied by nausea, and usually shows no tendency toward spontaneous recurrence. Similar morning emesis often accompanies alcoholism and excessive smoking. The rasping and scraping attempts to dislodge the secretions of the smoker's throat may continue until emesis occurs.

Occasionally in tuberculosis the vomiting does not depend upon the irritable condition of the throat, but may continue persistently, often for several weeks, the patient vomiting everything that is eaten during that time. In these cases, however, there is apt to be considerable temperature and the cause for the vomiting is often toxic.

Vomiting may occur in heart disease during the period of decompensation, either from congestion of the mucosa or from injudicious use of drugs, especially of digitalis. Anorexia and epigastric pain from hepatic engorgement complete the clinical picture.

Malarial paroxysms may be ushered in by vomiting and often with abdominal pain suggesting appendicitis. According to Cabot three such cases entered the Massachusetts General Hospital in a single week and were promptly cured by quinine.

When the above ordinary causes for vomiting and others which may occur to the observer, but which are not here considered, for obvious

reasons, have been excluded, there are found to be certain types of cases which may be included under the general heading of nervous vomiting, and which divide themselves into two groups, one accompanying organic disease of the nervous system, the other entirely of neurotic or psychoneurotic character.

Cerebral vomiting may occur in many lesions of the brain or membranes especially frequently with cerebral tumor. The patient will suddenly and without premeditation vomit from time to time without nausea or other manifest distress. The vomiting is apparently causeless and propulsive, and bears no relationship to the quantity or the quality of the food. Persistent and uncontrollable vomiting may usher in an attack of meningitis, especially of the tubercular form, and the vomiting may be so persistent and violent as to overshadow the other symptoms of meningeal origin. Attacks of severe periodical vomiting may occur, accompanied by severe headache, the ejecta consisting of acid fluid having all of the characteristics of acute periodical hypersecretion, or the gastroxynsis described by Rossbach. Unless the optic disk be examined, error in diagnosis may be committed.

Pain and vomiting occur periodically in tabetic patients constituting the well-known gastric crisis of locomotor ataxia. (See Gastric Crisis, p. 591.)

The occurrence of unexplained vomiting with great restlessness and incessant clamoring for relief without evidences of actual pain is rather characteristic of morphine habitués deprived of their drug.

Occasionally a paroxysm of vomiting is the sole manifestation of an attack of nephrolithiasis.

Vomiting with temperature should regularly suggest the onset of an infection, as vomiting in itself does not produce fever.

Hysterical or Nervous Vomiting.—Hysterical or nervous vomiting of a purely functional character may be seen in a variety of types, and is characterized in the main by the following peculiarities.

The vomiting act is causeless, unpremeditated, unaccompanied by nausea, and without much muscular effort. Vomiting during or shortly after meals is suggestive of the nervous origin, especially if the patient return to the table and eat again.

The vomiting shows no reasonable dependence upon the character of the food that is eaten. Simple food may be rejected while bizarre and unwholesome selections of food may be retained with comfort. One patient of the writer's could for a time eat nothing but chicken or lobster salad, while another subsisted for several days entirely on popcorn. Occasionally freakish patients are encountered who seem to possess the art of selective vomiting and can separate in some mysterious way what they wish to eject from the rest of the meal.

The vomiting is largely dependent upon nervous conditions of excitement, fatigue, emotional outbursts, or apprehension. Nervous vomiting may occur in students before their examinations or in lecturers or actors before their public appearance. The coexistence or alternation of other manifestations of a psychoneurosis are often strikingly apparent.

Despite even daily vomiting, loss of flesh and strength is quite exceptional, and there is often a strikingly disparity between the healthy appearance of the patient and the history of her having vomited after nearly every meal for weeks or even months. Increasing anemia and loss of weight should occasion doubt as to the purely neurotic nature of the complaint.

TYPES.—Three special types of nervous vomiting may be described:

Hysterical Type.—There is a type in which the patient will go day after day vomiting one or more times a day, occasionally after every meal, without nausea or distress, and without any apparent ill effects. It would seem as if the patient were capable by some miraculous power of existing indefinitely without food. It is probable, however, that only portions of each meal are rejected, leaving sufficient residue to sustain a fair degree of bodily health. Psychical vomiting may occur in normal individuals whenever subjected to shock, fright, or sudden mishap. If the cause for such an upset be repeated, a habit of vomiting may result even in the absence of a demonstrable cause. Chlorotic girls often vomit their food without any apparent reason for so doing. When, as is usual in such cases, the menses are suspended, the diagnosis from early pregnancy is quite difficult.

Erb's Juvenile Type.—Erb's juvenile type of vomiting is occasionally seen in susceptible, high-strung school children who overstudy and use up their nervous strength. The child may simply vomit from time to time without apparent cause, or the symptoms may come in attacks of headache, vomiting, dilated pupils, and occasionally a pulse rate somewhat slower than normal. These symptoms cease during the weeks of vacation or if the child be taken from school. It is questionable whether Erb's juvenile vomiting is a neurotic entity from overstudy or whether it does not result from some organic or reflex cause in impressionable overtrained children who are thus rendered unduly sensitive to such reflex stimulations. Eye-strain and masturbation should be suspected with such a clinical history, and the possibility of appendicitis should also be remembered.

Leyden's Periodical Type.—Leyden's periodical vomiting consists in vomiting attacks without apparent cause appearing at regular and orderly intervals. The attack begins with epigastric pain and vomiting, the ejecta consisting of what has recently been eaten, mucus and bile.

There may be an abolition of hydrochloric acid secretion, although gastric functions are usually normal. Even after the stomach has been emptied, repeated retching occurs, so that nothing is retained for any length of time. The patient shows the effects of the illness and becomes weak and prostrated. The attack may last several hours or may be prolonged for a week or ten days. The subsidence of the attack is usually abrupt, leaving the patient weak and exhausted but without other definite complaints. Recurrences occur after the lapse of several weeks or months, the distinctive feature being the absolute regularity with which the attack repeats itself.

The writer has not seen any instances that seemed to belong to this clinical type except those with almost identical symptoms that were evidently examples of the gastric crisis of tabes.

Cyclic Vomiting, Periodical Vomiting, Recurrent Vomiting.—Cyclic vomiting is a symptom-complex occurring mostly in children and characterized by periodical attacks of vomiting lasting from two to ten days, usually preceded and accompanied by marked constipation, together with acetonuria, indicanuria, and creatinuria.

Etiology.—The cause of this condition is unknown, but it is generally agreed that the disease occurs most frequently in children having an inherited or acquired neurotic constitution. Further, that it is in no way connected with dietetic errors.

Race, sex, climate, or season of the year play no part in its etiology. Family predisposition may be a factor, since several cases have occurred in the same family.

Theories as to causation abound. There are those who believe it to be closely associated with such other diseases as appendicitis, adenoids, constipation, hereditary gouty diathesis, rheumatism, and hepatic insufficiency. Others think it to be a pure neurosis or else connected in some obscure manner with the erythema group. All of the above find their basis in some clinical manifestation of the symptom complex. On the other hand a number of investigators believe that the disease has its origin in a deranged metabolism and present more or less conclusive proof for their belief. Experimental work has shown the following substances in abnormal quantities in the urine throughout the attack: acetone, diacetic acid, and beta-oxybutyric acid; uric acid, creatin, and neutral sulphates are increased at the beginning of the attack; lactic acid is occasionally found in severe cases; indican is increased greatly before and during the first days of the attack. According to Howland and Richards¹ all of the above may be traceable to a diminished power of oxidation of the tissue cells, hence they

¹ Arch. Pediat., 1907, p. 401.

believe that there is an interference with certain metabolic processes in which oxidation plays a part. They proved that when the oxidation power is diminished, indol acts as a poison. The accompanying constipation furnishes the indol which thus acts as a poison causing the intractable vomiting.

Sedgwick¹ believes that the metabolic disorder expresses itself in the constant presence of creatin in the urine, which becomes greatly increased just prior to an attack and gradually diminishes between attacks but never entirely disappears.

Mellamby² confirms the above findings and believes since creatin is always present that the cause of the disordered metabolism is always acting and that the accompanying acidosis, while not causative, is sufficient to produce the attack. He further believes that the underlying cause for the disordered metabolism is chargeable to some chemical product of bacterial action going on either in the intestinal mucosa or portal circulation.

Pathology.—There have been but few autopsies recorded. The pathological findings consist of varying degrees of degeneration of the gastric and intestinal mucosa. There is fatty infiltration of the liver, parenchymatous degeneration of the pancreas, spleen, and kidneys. Hypertrophy of the pyloric ring has been noted. X-ray plates taken near the end of attacks showed violent peristaltic waves passing across the gastric musculature.

Symptoms.—The attacks may occur at any time before puberty, most often beginning in the second or third year of life and continuing until the tenth year. At first the attack may take place eight or ten times a year with some regularity, but later occur at longer intervals and with less regularity. The attacks are ushered in with a period lasting two or three days in which lassitude, headache, abdominal discomfort and constipation are present. Distaste for food, later nausea and finally vomiting appear, or the vomiting may begin without any prodromal symptoms.

Usually there is throughout the attack a low grade of fever; generally constipation or obstipation; pulse may become very rapid and irregular; respirations may be greatly increased, irregular, and sighing. The odor of acetone usually permeates the sick room. Pain is frequently complained of but not definitely localized. Patients are at first very restless but later become exhausted and lie curled up in bed. There is intense thirst, the eyes become sunken, the body rapidly emaciates. The mind usually remains clear, but if the acidosis is extreme there may be marked stupor.

¹ Amer. Jour. Dis. Child., 1912, p. 209.

² Lancet, July 1, 1911, p. 8.

In mild cases the vomiting occurs spontaneously with but little retching. First the stomach contents are expelled, then the gastric juice and later the vomitus is composed of a bile-stained fluid. The vomiting may continue for two or three days and the attack then cease. The patient will then be perfectly well until another attack. In more severe cases the vomiting may occur every few minutes, accompanied by marked retching. If food be taken it is immediately expelled. The patient is unable to retain water. Owing to lack of nutrition and lack of water there is rapid decrease in weight. The muscles used in emesis become tender, the throat is inflamed, and the abdomen sunken. This may continue five or six days and the case assume a very serious aspect, when suddenly the vomiting ceases, the patient takes food and water, and convalescence proceeds rapidly. In the very severe cases the vomiting and retching completely exhaust the patient, the vomitus becoming at times streaked with blood or even distinctly bloody. There is marked emaciation, deeply sunken eyes, and frequently the patient enters into a state of semi-stupor, from which after protracted convalescence recovery may occur, or the case may go on to fatal termination. In these very few severe cases the child gradually sinks into coma and dies of the symptoms of acid intoxication in spite of all measures to counteract it. In others the fatal termination may be brought on by inanition and loss of water, together with the exhaustion due to the constant vomiting. Very few cases, however, result fatally. Nearly all convalesce rapidly, and, as a rule, are perfectly well until some exciting cause, such as fright, worry, fatigue, a railway journey, or some happening out of the ordinary brings on another attack. These gradually increase in severity up to the fifth year, when their intensity is diminished and finally about puberty disappear entirely, or they may continue in adult life as attacks of migraine.

Diagnosis.—This condition must be differentiated from indigestion, acute poisoning, migraine, meningitis, intestinal obstruction, peritonitis, and appendicitis. A history of previous attacks, together with the finding of acetone bodies in the urine, is of great importance in arriving at an early diagnosis. In the absence of history, together with the lack of signs of acute inflammation, one can temporarily rest a diagnosis on the finding of the acetone bodies. The diagnosis is confirmed when the attack ends suddenly and the acetone bodies disappear from the urine.

Prognosis.—A favorable prognosis may be made first as to recovery and secondly for complete cessation of attacks. Both parents and physician become more or less assured after having brought the patient through several similar attacks, and soon a prescribed routine is followed with favorable results.

Treatment.—In the matter of treatment, not knowing the cause, we have no specific cure. As a rule attacks come and go uninfluenced by medication. Prophylactic measures, such as removal of adenoids, tonsils, and appendix, circumcision, prevention of constipation, and measures to overcome neurotic tendencies, all have their advocates. Considering the attack as due to a deranged metabolism in which acid products are unneutralized, rational therapeutics would indicate an active alkaline treatment. Considering indol, skatol, and phenol as intestinal products whose toxicity is greatly increased, indications for their removal are apparent, hence a calomel purge followed later by high colon irrigations is often advisable. It is essential that the bowels be kept open freely throughout the attack. The marked loss of water from the system must be compensated for by water per mouth if possible; if not, by saline per rectum or subcutaneously. Lavage and gavage may be tried but are usually useless. Patients seldom can retain food in stomach, hence nutrient enemas rich in carbohydrates must be given. When vomiting threatens exhaustion, relief must be given by morphine. The usual antimitics have no effect. To break the habit is the main thing no matter what means are used. Cabot mentions an apparently sensitive workingman of thirty-five, who vomited continuously from habit until he had lost 55 pounds in weight, and was finally cured after a four months' duration of his vomiting by subpectoral infusions of saline solution.

MOTOR NEUROSES

Perigastric Unrest.—This term was used by Kussmaul to describe a peristalsis of the stomach, more or less continuously visible, not dependent upon pyloric obstruction. It is doubtful if it exists as a clinical entity of a pure neurosis.

Rumination.—Rumination consists in voluntarily bringing up food in small portions so that it may be either ejected or remasticated and again swallowed. If rumination occurs soon after meals the regurgitated portions of food may retain their original taste, but rumination later in the process of digestion brings up food either sour or bitter. Rumination is more common in men than in women, occurring only in 10 women of 145 instances reported by Presslich.¹ According to Elsner the habit is common in achylia and hypersecretion, but this cannot be verified by the writer.

The mechanism of rumination is imperfectly understood. It has been supposed that the habit depends upon relaxation of the cardia, but

¹ Wien. med. Woch., 1904, 17 to 21.

against this theory are the facts that the reswallowed food is retained without being again regurgitated and when the stomach is artificially inflated the air does not escape. The habit has been thought by some to be due to dilatation of the lower end of the esophagus and by others considered the result of irritation of the vagus, producing an opening of the cardia and antiperistalsis of the esophagus.

It has been thought that heredity plays a part in predisposing to the ailment, as in a number of instances several members of a family have been alike affected. It is more probable, however, that the habit is acquired by imitation, as an instance has been reported, for example, of a ruminating governess who was imitated by two of her pupils. The writer is more inclined to regard rumination as a faulty habit. The most marked example which he has ever seen occurred in a young man who was imprisoned in Russia for a political offence, and who contracted the habit because his food was so scanty that it was only by rumination that he could sufficiently protract his meal. This individual had the power of selection, so that after a mixed meal he could separate any one article of food and bring it up for remastication.

The treatment of rumination is that of an evil habit, by autosuppression and psychotherapy. The patient should be forced to eat slowly and to masticate thoroughly. Strychnine and quinine are of service only because of their bitter taste. They remind the patient that he is ruminating. Intragastric galvanism with the descending current has been employed with striking results, probably the result of therapeutic suggestion.

Hypermotility.—Hypermotility is a term designating an acceleration of food exit from the stomach so that the organ is empty before the normal time. The condition may occur occasionally as a temporary motor neurosis but is not of any clinical importance. Hypermotility is said to be common with achylia. This is true in the writer's experience in a few but not in the majority of instances. Rapid evacuation of the stomach in the absence of the acid reaction required to open the pyloric gate is a problem not easily explained. It is claimed that hypermotility is common with duodenal ulcer, as fluoroscopy shows rapid entrance of the bismuth meal into the duodenum.

Pyloric Insufficiency.—This may ensue from organic lesions of the pylorus, such as cicatrizing ulcer or cancer, which produces a rigid patency of the orifice, which acts in two ways. The opening is not sufficient for the easy passage of food from the stomach to the duodenum and at the same time cannot contract sufficiently to prevent duodenal regurgitation. The condition may be recognized by finding food remains in the fasting state, together with bile and other contents of the small intestines.

Pyloric insufficiency from neurotic causes is extremely rare. The condition may be surmised whenever air introduced into the stomach for the purpose of inflation passes into the intestine simultaneously with a rapid deflation of the stomach itself, or when in apparently normal individuals bile and pancreatic juice are repeatedly present in the fasting stomach. After the patient is thoroughly accustomed to the passage of the tube, the condition is difficult to be distinguished from the duodenal regurgitation following an excessively fatty diet, and may occasionally be indistinguishable from the duodenal distention that results from duodenojejunal kinks.

SECRETORY NEUROSES

Disorders of secretion, either consisting of subacidity or hyperacidity, may be of nervous or functional origin. Hyperacidity was apparently a nervous phenomenon in 20 per cent. of the writer's series, or to state it more accurately, in 20 per cent. of the writer's cases of hyperacidity no organic cause could be ascertained. This does not necessarily imply, however, that organic disease did not exist. In functional hyperacidity neither heart-burn nor pain seem to occur, nor was vomiting a feature of the ailment, so that the presence of any one of these symptoms would suggest organic origin for the hyperchlorhydria. The subject is discussed in full under Hyperacidity, page 463.

Achylia may undoubtedly be due to a nervous inhibition of secretion. It is a matter of common experience that a meal eaten under great nervous excitement or fatigue may be vomited within a few hours completely undigested and show no trace of gastric juice.

Temporary achylia may also precede menstruation or may be noted during the first day or two of the period. These instances of nervous achylia are of short duration, the flow of gastric juice being normally established whenever the nervous system becomes again tranquillized.

In other instances achylia may appear as a permanent condition without apparent cause in those who are otherwise healthy in every particular. One-third of the writer's cases of achylia occurred in those in whom a physical examination was absolutely negative. It is interesting to note, however, that with gastropotosis a condition almost invariably accompanied by neurasthenia more or less profound, achylia has not occurred in the writer's series more frequently than in a similar number of patients not so afflicted.

Gastric symptoms with achylia should suggest lesion in the gall-bladder or appendix or the possibility of an arteriosclerotic origin.

Achylia with food-stasis should suggest cancer. A discussion on the various aspects and causes of achylia is given on page 482.

Instability of gastric secretion is observed in some neurotic patients and is demonstrated by fluctuations of acidity without apparent reason in the same individual. An example of this so-called "heterochylia" is that of a patient of the writer's, a nervous woman, profoundly depressed by domestic infelicity, in whom the test breakfast on one day was of a total acidity of 78, the following morning of 6. In another patient suffering from petit mal, achylia was present one day, while on the following morning the test breakfast showed an acidity of 136. Heterochylia affords corroborative evidence of a neurosis, but as it may coexist with organic disease it is of less value in diagnosis than one would imagine. *The greatest lesson to be learned from the symptom is that more than one gastric analysis is necessary for the complete study of a given case.*

Continuous hypersecretion indicates a rudimentary form of pyloric stenosis, either spasmodic or organic, and cannot therefore be regarded as evidence of a neurosis. Alimentary hypersecretion, according to the German school, occurs in nervous individuals who present the symptoms of nervous indigestion. This is, however, contrary to the writer's experience.

TREATMENT OF GASTRIC NEUROSES

Tact, consideration, and real, not maudlin, sympathy are required. A mental attitude of boredom when called on to treat nervous, whimsical, and hypochondriacal patients is fatal to good results. Failure to relieve our patients is often due to the negligent and casual way with which we treat them, and to our utter lack of human sympathy. Their complaints may not be very interesting oftentimes to us, but to them they are real, vivid, and harassing, and their mental distress may be as devitalizing as actual physical pain. Nervous indigestion is after all a habit neurosis in many instances, and the only way to permanently check the habit is by reëducation of the nervous centres.

The treatment is that practically of suggestion, of quieting undue fears and apprehensions, arousing ambitions and self-help, and inspiring the patient with a sense of his own power of helping himself.

We may or may not be able to change environment. Time brings healing on its wings after nervous shocks and vicissitudes of fate, but the difficult problem is to deal successfully with those that are temperamentally unfitted for the place in life in which they have to remain. Especially is this the case with those afflicted with the enteroptotic habitus who are small capitalists, easily bankrupt in every nervous panic.

Rest, travel, and freedom from care and worry are all desirable and may be carried out with more or less completeness, according to the circumstances of the given case. Daily routine occupation often takes patients out of themselves, so that many are better for hard and engrossing work. There should in all cases be a proper equilibrium between nervous outgo and nervous income. Work and activity should be balanced by corresponding periods of rest and recreation. In many instances absolute rest for a certain period in the afternoon should be encouraged. In those who are debilitated, especially those with visceral ptoses, a rest cure is often essential for their restoration to health.

It is of importance to see that the patients eat enough. Their natural tendency is to be unduly apprehensive and to attribute their distress to the food that they have recently eaten, so they cut down their diet and restrict themselves.

The treatment is essentially irrational when the victim of nervous indigestion is allowed continually to lose weight. Another natural tendency is for the patients to overmedicate themselves and to be constantly taking headache powders, desepsia tablets, and any remedy that may be suggested by sympathetic but not always intelligent friends. Bromides may be of service for short periods of time to tide over exacerbations of the ailment, but this treatment should not be long continued for reasons that are quite obvious.

Treatment of Special Symptoms.—Anorexia.—A change of scene is often desirable, especially for nervous housekeepers who have to plan ahead for their meals. Food taken in solitude is unappetizing compared with that enlivened by pleasant associates.

Drugs are of little service. Menthol may be given in half-grain doses, either in capsules or in the following prescription before meals:

R—Menthol	gr. xvi
Alcohol	℥ss
Syrup. simplicis	ad ℥iv

M. Sig.—Teaspoonful in water before meals.

Orexin tannate in 5-grain doses has been recommended, but in the writer's experience has been practically worthless. A common form of treatment is that of bitters with or without *nux vomica*, and is perhaps the most reliable of all medicinal treatments.

The following prescriptions may prove of service:

R—Tinct. nucis vomicæ	℥v
Elixir condurango	ad ℥iv

M. Sig.—Teaspoonful in water before eating.

R—Tinct. physostigma	℥iv
Tinct. chamomilla comp.	ad ℥iv

M. Sig.—Teaspoonful in water before meals.

Achylia.—If achylia be present, dilute hydrochloric acid or oxyntin may be given during or after the meals. It is important to see that the patient does not drink too much water between his meals so that he is producing an artificial plethora which effectually reduces his desire for food.

Bulimia.—Bulimia is to be combated by mental suggestion. Drugs are of no avail in this condition. The patient may be advised, however, to carry chocolate tablets in his pocket so as to relieve the craving for food.

Hyperesthesia.—Hyperesthesia is to be treated by attention toward the underlying condition. In gouty subjects the treatment should be directed toward this diathesis. An experimental treatment by colchicum is often of service in establishing the diagnosis. When hyperesthesia is a reflex manifestation of gallstones or of gall-bladder infection, treatment toward the latter condition is indicated. A course of Carlsbad water, either the imported or the artificial, should be given as hot as can be sipped the first thing in the morning, and one-half such a dose on retiring, limiting the quantity, however, so that not more than the liquid or two uniform bowel evacuations will result. Ten grains of sodium salicylate in hot water may be given before breakfast and before dinner, or 2 tablets, $7\frac{1}{2}$ grains each, of sulpholythin with hexamethylenamine (Laine Chemical Co.) may be given with a glass of hot water a half-hour before each meal.

Gastralgia.—The principles of treatment are quite evident from the consideration of what has been said concerning this symptom. Chlorotic girls should be treated by iron and saline laxatives. Syphilitic patients should receive appropriate treatment. The possibility of malarial infection must be borne in mind. As gastralgia is of rare occurrence as a pure neurosis the treatment of pain in the stomach is naturally that of the underlying organic disorder to which the pain is due.

Vomiting.—The treatment of nervous vomiting is a thankless task, although more results are to be expected from general treatment than from medication of a more purely local character. A variety of anti-emetics may be used, such as resorcin, menthol, drop doses of carbolic acid, or of diluted hydrocyanic acid, but the effect produced is temporary and the disorder speedily recurs. The treatment is generally unsatisfactory unless a definite cause for the vomiting can be discovered.

Nervous Eructations.—The treatment concerns itself largely with the correction of the underlying neurosis and is usually a thankless task. Instances have been reported of rapid and complete cure after the nature of the ailment has been explained to the patient, but in the writer's experience explanations as lucid and convincing as lay in his power have not influenced in the least the severity of the complaint.

Psychotherapy and autosuggestion should, however, constitute an important part of the treatment. Mitigation of the distress by drugs is often a thankless task. Bromides, valerian, and chloroform water may be employed—belladonna may be given to the point of mild physiological intolerance, and while the results may for a time be apparently encouraging, and results are usually quite disappointing, while in many cases not the least effect can be apparently produced.

Should the eructations recur so rapidly that the patient is becoming worn-out, temporary relief follows the placing of a cork between the teeth, thereby impeding or preventing the pharyngeal spasm. A similar effect is produced by passing a stomach-tube and retaining it in position for a quarter to a half-hour at a time, with the additional advantage of wearing out the spasm.

CHAPTER XXI

VARIOUS DISEASES AND THEIR GASTRIC RELATIONS

APPENDICITIS

Forms.—Appendicitis, both acute and chronic, is a frequent cause for upper abdominal pain.

Acute Appendicitis.—Acute appendicitis often begins by severe epigastric pain and vomiting. The diagnosis often made of acute gastritis is always unjustifiable when pain is present. Acute painful “gastritis” should suggest ulcer, acute appendicitis, or acute hypersecretion. The latter condition can be readily excluded if the vomited matters do not consist of acid liquid. A downward radiation of the pain is suggestive of acute appendicitis, as such a distribution is not observed with ulcer. The pain gradually moves downward to the middle abdominal zone and finally centres in the right iliac fossa, making certain the diagnosis.

A polynucleosis may be of service in the diagnosis if present during the early stage of the complaint.

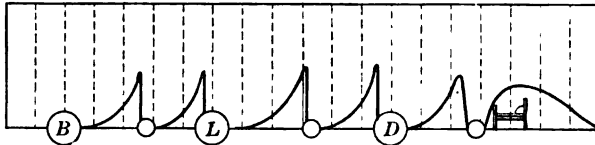
Chronic Appendicitis.—Chronic appendicitis is responsible for many errors in diagnosis, as the disease is capable of generating a great variety of gastric complaints without revealing itself either by the history of previous attacks of inflammation or by pain or tenderness in the right iliac fossa, even though repeated careful examinations be made. The diagnosis, therefore, in many cases is arrived at by a process of exclusion and the appendix removed on the supposition that it is the *fons et origo mali* without being clinically convicted of guilt.

Symptoms.—The writer does not intend to describe the symptoms of chronic appendicitis in full detail, but merely to speak of the gastric symptoms which such a condition may produce and which may be confused with local disorders of the stomach. The gastric symptoms of chronic appendicitis may be divided into four clinical groups.

I. Pain Type.—Bilious attacks in children with pain, headache, and vomiting, usually ascribed to an unwholesome meal, frequently culminate in an obvious attack of appendicitis. After operation the bilious attacks cease. In those on whom no operation is performed the symptoms of these early attacks may continue through childhood and gradually merge into those that closely resemble gastric ulcer. Pain may occur two or three hours after eating and be relieved by eating, as in

ulcer. The cause for such recurring distress is a protective spasm of the pyloric sphincter, which is of itself sufficient cause for pain. A

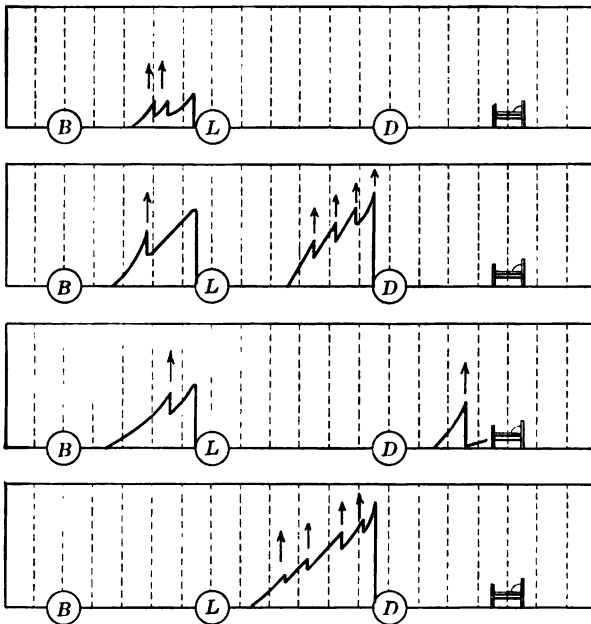
FIG. 117



Pain line in a case of chronic appendicitis, with hunger pain relieved by eating. Cured by appendectomy. (Circles represent meals.)

mild form of chronic hypersecretion almost regularly accompanies the paroxysm and is an added factor in increasing the severity of the distress.

FIG. 118



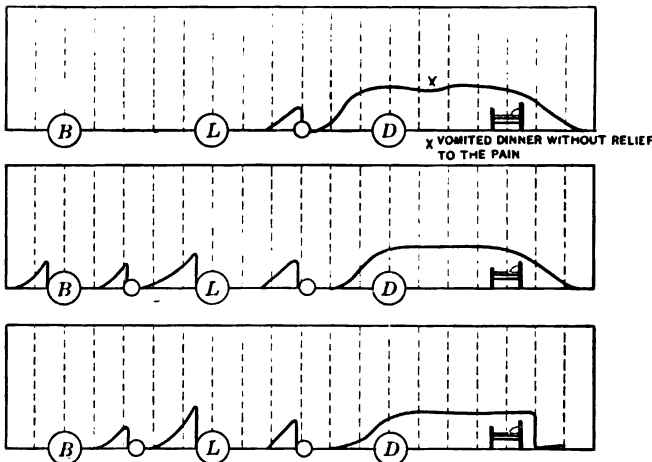
Pain curve of a case of uncomplicated chronic appendicitis cured by operation. The pain line conforms to that of the hunger pain. The arrows indicate eructations of gas which are followed by temporary relief.

In some instances the feeling is one of great distress temporarily relieved by raising gas—more completely relieved by eating.

Pain in the stomach occurs after eating, as in ulcer, but the time of its appearance after the meal is less definite than in the latter disease

and lacks its clock-like regularity. The pain may thus appear on one day an hour after meals and on other days two or three hours after food has been taken. Its capricious appearance is recognized by the patients, who often say their pain comes at any time. This irregularity of the pain after the meals is of value in differential diagnosis, but unfortunately in many instances the pain appears with the same definite regularity after eating as does the pain of ulcer, so that a differential diagnosis may be utterly impossible.

FIG. 119



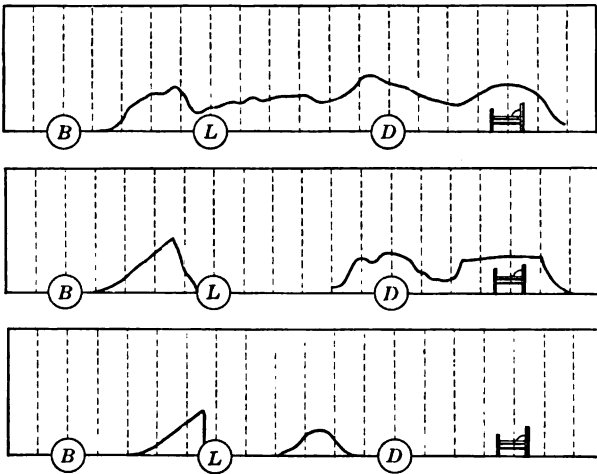
Pain curve of an uncomplicated case of chronic appendicitis cured by operation. The pain during the day is of the hunger type, relieved by eating. (The smaller circles indicate nourishment given between the regular meals.) The evening pain is unrelieved by eating or vomiting, ceasing only by rest. This evening type of pain is not uncommon with appendicitis, rare with ulcer.

It is suggestive of appendicular gastralgia when exertion or hard physical work increases the severity of a pain which food has caused. It is also suggestive of the appendicular origin of the pain that relief afforded by food, drink, or alkalis is less immediate and complete than in ulcer. In some instances the pain has no definite relationship whatever to the taking of food, but is more or less continuous, lasting for two or three days without intermission, although at no time very severe, and then ceasing until it recurs with a succeeding attack.

The location of the pain roughly speaking is epigastric, but lacks the accurate localization that is seen in ulcer. In many cases the pain is felt lower down in the abdomen below or to the right of the navel, and even though the pain may originate in the epigastrium radiation downward toward the umbilicus or lower abdomen may occur. This radiation seldom, if ever, occurs with ulcer or disease of the gall-bladder.

One of the most practical rules in distinguishing between ulcer and the appendicular form of indigestion is afforded by the behavior of the patient with supposed ulcer during his ulcer cure. If after two weeks of treatment the original symptoms persist, even though they be in a modified and less severe form, chronic appendicitis must be considered possible. In many cases, however, a differential diagnosis cannot be made—only operation can decide.

FIG. 123



Pain chart of a case of acute exacerbation of chronic appendicitis. The pain on the first day is practically continuous and unrelieved by eating. The pain on the second day is practically unchanged by eating dinner. The morning pain on the third day is that of the hunger type.

The following history shows how closely the clinical course of appendicitis approaches that of ulcer.

ILLUSTRATIVE CASE.—G. S. M., male, aged thirty-four years, entered the hospital with the following history: He had always been free from indigestion and from all abdominal distress until a year ago, when he began to suffer from pain in the pit of the stomach between two and three hours after eating, lasting until he ate again. Instant relief followed the ingestion of food, and the larger the meal the longer the period of comfort that succeeded. These symptoms have persisted with great regularity since the onset, so that he has not been free from pain for a single day. Spontaneous vomiting has not occurred, but he has often induced emesis to relieve himself of pain, and almost invariably with success.

Physical examination: Stomach is apparently of normal shape and size. There is a markedly tender point localized just over the ensiform. Head's hyperesthetic zone is well-marked, terminating behind in a

distinct dorsal point of tenderness. Repeated examinations showed no tenderness in the right iliac fossa. Fasting stomach contained 25 c.c. of clear fluid without food remains. Total acidity 60, free hydrochloric acid 45. Test breakfast: 160 c.c. well-digested breadstuff, separating on standing into two layers, the supernatant layer being two and a half times the depth of the sedimentary layer. Total acidity 98, free hydrochloric acid 60, slight trace of occult blood present.

Patient was placed upon the von Leube ulcer cure for four weeks. The symptoms improved markedly after the fourth day, but did not entirely disappear, and at times he would suffer from a mild discomfort two or three hours after eating. This discomfort was, however, so insignificant that he regarded himself as cured. The tenderness in the epigastrium completely disappeared. After he had been up and around the ward for several weeks he suddenly complained of intense, agonizing pain in the epigastrium, radiating thence over the whole of the abdomen, and passed into a condition of surgical shock. The abdominal wall was rigid and board-like, his temperature subnormal, his pulse rapid and feeble. Exploration for a supposed perforation of a duodenal ulcer was done two hours after onset of pain. The stomach was normal, inside and outside, as was the duodenum. A perforation of the appendix had occurred from acute gangrene of its tip without adhesions. Recovery from the operation was uneventful, and for the following two years, during which time the patient was under observation, he had no gastric distress of any kind, but could eat all sorts of food with impunity.

II. Nausea Type.—A persistent nagging nausea may be the sole symptom of appendicular disease. The nausea is seldom pronounced, but is of low-grade intensity, characterized by its constancy rather than its severity. It comes and goes throughout the day, sometimes before meals and other times after meals, and having no fixed time for its appearance. It does not seem to interfere with a reasonable enjoyment of food, nor is it made worse by eating. Such a history is as follows:

Mrs. L., aged forty-three years, for six or seven years has been nauseated every day, although seldom to the point of vomiting. Despite the nausea she has been able to eat and to digest her food with comfort. All forms of treatment, including anchoring the kidney, have produced no beneficial result whatever. For the past two months she has complained of an occasional pain in the stomach, not due to gas, appearing about two hours after eating and lasting until she has eaten again. One week ago the definite symptom of an acute attack of appendicitis appeared. Operation totally relieved her, not only of her recurring epigastric pain but of her chronic nausea.

Cases of this type are often considered to be instances of nervous indigestion. A feeling closely allied to that of nausea is the sense of disagreeable stomach emptiness after eating, suggesting to the patient that he eat again. This symptom, described by Boas as a neurosis (see *Gastralgokenosis*), has occurred in a number of cases of chronic appendicitis observed by the writer and has been relieved by operation.

III. Vomiting Type.—Recurring acute attacks of appendicitis or acute exacerbations of a chronic inflammation may cause vomiting for a period of several days at a time, the vomited matters consisting either of food recently taken or of acid fluid characteristic of hypersecretion. The amount of acid fluid is not as copious as with ulcer. Pain usually accompanies these attacks, having a tendency to radiate downward and to be more or less continuous and uninfluenced to any great extent by the taking of food.

Vomiting with appendicular indigestion does not bring the same degree of relief to the patient as does the vomiting of ulcer.

There is a type of chronic appendicitis characterized by daily vomiting continued over long periods of time without apparent cause. Vomiting may occur once or twice a day only, or may be repeated after every meal, even though the symptom extend over months or years. In some cases food is ejected soon after eating without accompanying nausea, so that the patient after an attack of emesis is quite willing to eat again. Vomiting of the entire meal is rarely observed, so that the patient usually continues to be of good nutrition and to appear outwardly healthy.

In other cases the vomiting is quite erratic and bears no relation to the meals. There may be intermissions, often of several months or more. In one of the writer's cases an intermission of two years occurred. Sooner or later, however, the symptoms return and cease permanently only when the appendix is removed. Medical treatment is of no avail in relieving the distressing symptoms.

Patients of this group are regularly supposed to be suffering from nervous indigestion.

ILLUSTRATIVE CASE.—A history of such a patient is as follows:

Mrs. F., aged thirty-seven years, was well until seven years ago, when after a nervous upset she began to suffer from repeated attacks of vomiting. At first vomiting occurred only when she was more than ordinarily nervous and would cease when her equanimity was restored, but after two or three years she began to vomit after every meal and has continued to do so until the present time. The vomiting is largely under her control, so that she can postpone the emesis until a favorable opportunity occurs. It has not been influenced by diet, lavage, or any medical treatment whatever. Physical examination and gastric analysis were

normal. At operation a chronic obliterating appendix was removed, followed by an abrupt and permanent cessation of all previous symptoms.

Vomiting of blood is not uncommon, occurring in 5 out of 24 cases reported by Paterson. In 12 cases in Moynihan's series the patient vomited over a pint of blood at one time. The combination of epigastric pain and hematemesis so closely resemble ulcer in many instances as to satisfy the most exact clinician. There has been much discussion as to the cause for the bleeding in these cases. The hemorrhage has been ascribed to sepsis, to embolism of an artery of the stomach secondary to thrombosis in the omental branches, or to toxic conditions which are at present obscure.

Some observers attribute the bleeding to an oozing or a weeping of blood from the entire mucous membrane of the stomach without actual loss of continuity. The writer believes that many of the hemorrhages are due to traumatism of the mucous membrane of the pyloric canal caused by the spasm, which results in a lack of vitality of tissue and the production of minute superficial erosions due to self-digestion.

IV. Gas Type.—There is an important group of cases whose sole complaint is that of gas in the stomach two or three hours after eating, giving rise to discomfort or even to pain, until relieved by free eructations. These symptoms may appear in short attacks or the distress may be attended with fluctuations in its severity and may be prolonged for several days. There is a lack of correspondence between the sense of distention which the patient experiences and the actual inflation of the stomach by gas, as determined by the physical examination. Distress from flatulence is more severe and continuous in appendix dyspepsia than in the ulcer and is more apt to be general and unlocalized. It is, furthermore, suggestive that the raising of a very small amount of gas will bring for the time being a complete relief. The distress seems rather due to hypertonus of the stomach than to an actual distention of the viscus by gas. The history of such a case is as follows:

Mrs. A. M., aged twenty-eight years. A nervous woman and subject to temporary upsets of her digestion from time to time, was comparatively well until three years ago, when she began to complain for several weeks at a time of gas in her stomach several hours after eating, giving her severe cramp-like pains which radiated upward to the left shoulder. This was her only complaint.

Physical examination showed slight tenderness over McBurney's point. Result of gastric analyses inconclusive. Uneventful recovery followed appendectomy without recurrence of the symptoms in the five years that have elapsed since her operation.

Diagnosis.—It is unfortunate that in the majority of instances the examination of the fasting stomach and of the test breakfast in chronic appendicitis shows no abnormalities whatever. The accompanying table gives the total acidity of the test breakfast in 100 cases of uncomplicated chronic appendicitis:

Total acidity 1 to 10	4 cases
Total acidity 11 to 20	3 cases
Total acidity 21 to 30	9 cases
Total acidity 31 to 40	16 cases
Total acidity 41 to 50	22 cases
Total acidity 51 to 60	23 cases
Total acidity 61 to 70	15 cases
Total acidity 71 to 80	4 cases
Total acidity 81 to 90	3 cases
Total acidity 91 to 100	1 case
	<hr/> 100 cases

It will be seen by this table that hyperacidity of over 70 was present in but 8 per cent. and that anacidity (acidity under 20) was present in but 7 per cent. These are about the proportions seen in the average run of patients with indigestion as they come consecutively under observation.

Achylia, for example, occurs in about 6 per cent. of all patients. With chronic appendicitis it occurs in 7 per cent., showing there is no direct connection between these two conditions. The writer has found a hyperacidity of 70 and over in 13.8 per cent. of all patients suffering from digestive disorders, while this complication occurred in 8 per cent. of the cases of chronic appendicitis, a difference quite small though probably suggestive.

More important, however, is the examination of the fasting stomach as an indicator of chronic hypersecretion. In 12 per cent. of the writer's cases of chronic appendicitis, quantities of gastric juice of 30 c.c. or more were found in the fasting state. Amounts under 30 c.c. have been disregarded.

Between 31 and 40 c.c.	7 cases
Between 41 and 50 c.c.	2 cases
Between 51 and 60 c.c.	0 cases
Between 61 and 70 c.c.	1 case
Between 71 and 80 c.c.	1 case
Between 81 and 90 c.c.	1 case

As this quantity (30 c.c.) was found in a trifle less than 4 per cent. of all patients suffering from digestive disorders, it is, therefore, evident

that mild hypersecretion is three times more frequent with chronic appendicitis than in the general run of dyspeptic patients.

Physical Signs.—Physical signs may be totally lacking. Repeated examinations of the region of the appendix may show at no time the slightest degree of tenderness. If tenderness be present it is usually rendered more evident by palpating McBurney's point after the colon has been moderately inflated by the rectal injection of air. Tenderness over the appendix was noted in one-third of Paterson's cases. If one is content with but a single examination, the writer believes that the number of cases of appendicitis without tenderness is far less than this. Repeated examinations may, however, at some particular time elicit a tenderness over the appendix that suggests the correct diagnosis. Tenderness in the epigastrium, usually somewhat to the right of the median line, occasionally as far down as the level of the umbilicus, occurs almost constantly, but is not as sharply located as is the tenderness with ulcer.

The course of the disease is progressive with periodical exacerbations. During the interval of the attacks more or less discomfort and flatulence are present, so that at no time does the patient really feel well.

Treatment.—There is no medical treatment for appendicular indigestion that is of much benefit. The symptoms may be temporarily relieved by the bland diet employed in the convalescence of the ulcer cure, or by prolonged rest in bed, but the relief thus afforded is merely temporary. It is the persistence of the symptoms despite our best medical care of the patient that often furnishes us with the correct interpretation of the case. Operation, therefore, is to be advised. Ordinarily it is the chronic obliterating appendix that gives rise to the gastric symptoms, but this is not the form which easily perforates. The operation, therefore, is not one of urgency, although it should be performed without unnecessary waste of time. The stomach and gall-bladder should regularly be explored at the time of the operation, unless there are definite reasons why this should not be done.

DISEASE OF THE GALL-BLADDER AND GALLSTONES

In many instances gallstones run a latent and symptomless course and are found at operation in cases which have presented no clinical evidence of their presence. In other cases the symptoms are purely local, or at least the local symptoms so predominate the clinical picture that no difficulty is experienced in arriving at the correct diagnosis. In still other cases the local manifestations of the disease may be

slight, often so insignificant as to elude observation, while the bulk of the symptoms are those of apparently gastric origin.

Forms.—The writer will only allude to those forms of gall-bladder disease or gallstones which present the picture of gastric indigestion.

1. The passage of a stone into the cystic or common duct results in immediate, severe, and lancinating pain, which, suddenly appearing in the epigastrium, radiates to the right and upward or to the right side of the back. Radiation to the left may occur, but is quite unusual. The pain is continuous, with periods of intense exacerbation, often to an almost unendurable degree, and is uninfluenced by food, fluids, or alkalies. There may be a distended bursting feeling due rather to the character of the pain than to gas. Vomiting frequently occurs but affords no relief to the agony, as in ulcer. The ejecta consist of recently ingested food, bile, and mucus, often of acid reaction, often the result of repeated and painful retching. According to W. J. Mayo the vomitus more often contains pure bile than does the material vomited in cases which might be mistaken for gallstone disease.

Relief is only obtained by sufficient doses of morphine or by the natural termination of the attack. Should the stone drop back into the gall-bladder or pass the common duct, the cessation of pain is abrupt; but should the stone become impacted in either the cystic or common duct, the pain may temporarily subside, only to be renewed with every fresh excursion of the concretion.

Jaundice does not occur with stone in the cystic duct unless it be impacted near the junction and press upon and occlude the common duct. Should the stone engage in the common duct or should the bile ducts become inflamed jaundice may result. Less frequently jaundice may occur from adhesions and kinking of the common duct, or by compression of the ampulla by swelling of the head of the pancreas.

During the attack there may be local rigidity of the upper portion of the right rectus and of the right costal arch, with tenderness in the region of the gall-bladder. When these signs are present the diagnosis is quite evident.

2. Should the stone engage in the cystic duct the pain is frequently neither severe nor prolonged, but occurs in mild and repeated paroxysms—a clinical course quite indistinguishable from that of acute cholecystitis next to be described.

3. Distention of the gall-bladder may result from the impaction of a small stone in the cystic duct, or may be due to acute cholecystitis, or to an acute exacerbation of the more chronic form of infection, which results in the swelling of the orifice or blocking the passage into the duct by mucus. The attack begins with an uneasy feeling in the stomach, as if wind were there that should be raised, although there may be no

demonstrable inflation by the physical examination at the time. The patient will take hot water, soda, alkalies, and aromatics without relief. There may be some gas raised but the quantity is insignificant and does not mitigate the distress. The pain then usually becomes of a dull and aching character, often described as "grinding," and is nagging and persistent, and not capable of relief in any way except by morphine or codeia. These symptoms seem regularly due to gastric spasm reflexly induced by the irritative lesion in the gall-bladder. Vomiting may occur, the ejecta consisting mainly of recently ingested food without definite characteristics.

4. In other cases painful gaseous distention may occur from time to time without relation to meals or other apparent cause. Relief is regularly afforded for the time being by carminatives, which succeed in raising the gas in large quantities, with immediate relief. It is rather characteristic for these paroxysms to occur during the early part of the night, so that the patient will sit up for several hours attempting in various ways to raise the gas and to procure relief. Many cases of flatulent dyspepsia seen in dispensaries belong to this group of cases.

5. There is a close association between gall-bladder disease and achylia. Twenty-two per cent. of the writer's cases of achylia were associated with gall-bladder disease or gallstones, while in the gall-bladder cases achylia was present in 30 per cent. The functional forms of achylia and those due to chronic gastric catarrh run a painless course. Achylia accompanied by pain is regularly indicative of an organic complication. Especially should be considered in these cases gastric cancer, gall-bladder disease, chronic appendicitis, and angina abdominalis.

6. Of the writer's series of gall-bladder and gallstone cases hyperacidity was found in 30 per cent. Functional hyperacidity is not accompanied by either heart-burn or pain, and the occurrence of either of these symptoms should suggest an organic origin, especially ulcer, cancer, gall-bladder disease, or chronic appendicitis.

7. With disease of the gall-bladder and with gallstones a protective spasm of the pylorus may be induced, resulting in recurring epigastric pain or in the symptoms of hypersecretion. There may be thus a close mimicry between gastric ulcer, gallstones, and chronic appendicitis, so that in a doubtful case it is well not to express too positive an opinion. The diagnosis is rendered still more difficult by the possibility of two or all of these conditions being associated in the same patient. No surgical exploration performed for the sake of determining the origin of recurring epigastric pain is complete unless the stomach, duodenum, appendix, and gall-bladder be explored.

ARTERIOSCLEROSIS

Arteriosclerosis is of common occurrence in those of advancing years as well as in younger subjects in whom the combination of alcohol and hard work, gout, lead poisoning, syphilis, and various toxemias from infectious disease, errors in metabolism, and toxic absorption from intestinal stasis, may be demonstrated to be predisposing or precipitating factors. In other patients the arterial disorder may appear as an evidence of premature senility or as the result of causes at present obscure. It is not within the scope of this section to deal with the causes of or the symptoms produced by arterial disease in general, but to confine the discussion to the various conditions of the stomach produced by such a malady, and to describe the symptoms by which an arteriosclerotic origin of the complaint may be recognized or suspected.

Etiology.—There seems to be but little relationship between the development of the arterial disease on the one hand and the symptoms which it produces on the other. Gastric symptoms may be present in cases in which the arteries are apparently but slightly involved and totally lacking in the advanced forms of the disease so commonly seen in our hospital wards.

In many of the cases that are apparently latent as far as symptoms of indigestion are concerned, it is probable that atrophy of the mucosa with an accompanying chronic interstitial inflammation is present without producing symptoms of any indigestion whatever. Achylia occurs in 60 per cent. of patients over fifty, whether symptoms of gastric disorder are present or not. The changes, productive and atrophic, in the gastric mucosa are more probably lesions associated with arterial sclerosis than directly due to it. The only suggestive symptom produced by this phase of the disorder is morning diarrhea. A full description of these cases is given under the heading of Achylia, page 496.

Local thickening of the wall of any of the gastric arteries may lead to blocking of the blood supply of a portion of the stomach, so that the affected area is eroded by gastric digestion and ulceration results. This intoward event would occur far more frequently than it does were it not for the tendency toward achylia as age advances.

Miliary aneurysms produced by local atheromatous degeneration may occur in the course of any of the gastric arteries and may lead to sudden and usually fatal hematemesis. The production of the aneurysm is favored by ulceration of the overlying portion of the mucosa. The peptic ulcer may or may not have given previous symptoms of its presence. With the history of preceding pain, followed by profuse

hemorrhage, the diagnosis from the ordinary chronic ulcer and erosion of a branch of the gastric artery is quite impossible. If the previous history of ulcer cannot be elicited, a differential diagnosis from rupture of esophageal varices, the result of cirrhosis of the liver, is often difficult, especially as cirrhotic patients are subject by reason of alcohol and their mode of living to widespread arterial disease.

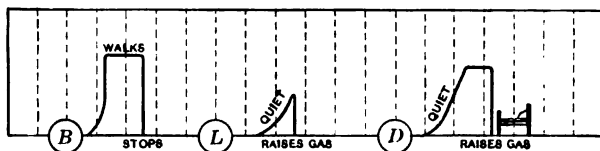
An arteriosclerotic ulcer may be suspected if, preceding the hemorrhage, there be the history of epigastric pain or sudden attacks of flatulence, occurring whenever the patient walks after his meal and which do not occur if he rests after eating.

In advanced arteriosclerosis with signs of failing heart and venous congestion, anorexia, vomiting, and the pain of hepatic engorgement may occur. These are the symptoms common to all cardiac cases in the stage of decompensation.

In other patients gastric symptoms may appear for a considerable time before characteristic symptoms of arteriosclerosis develop, and may be divided into two groups, according to whether flatulence or pain constitutes the predominant symptom.

Symptoms.—Gas Type.—Attacks of flatulence may occur regularly after eating, appearing usually at the height of digestion with some abruptness and subsiding gradually as gastric digestion wanes. In some instances the attack is abruptly terminated by the eructations of gas which instantly relieve the distress. The symptoms do not appear with every meal and for days there may be freedom from the attacks.

FIG. 121



Pain chart of a patient with angina abdominalis, showing two types of pain, one type coming on while walking and instantly relieved by stopping, the other, appearing during the height of gastric digestion, relieved by raising gas.

The paroxysm may be precipitated by exercise, especially walking after the meal. The gas is often in large amounts and produces as much oppression in the chest as in the abdomen. This oppression is often so extreme that the patient has to stop whatever he is doing until he can raise the gas, after which he is not apt to suffer until after some succeeding meal. The attacks are not apparently influenced in the least by the character of the food that is eaten. Nervousness almost regularly makes the gas worse. In other instances the attacks occur whenever the patient walks after eating. If he sits quietly or rests he is free

from all distress, but upon moving around, walking or undressing, or occasionally induced by changes in temperature, an attack quickly supervenes. There is in the majority of the cases the background or arteriosclerosis in some more or less characteristic form.

ILLUSTRATIVE CASES.—The following histories may be cited as examples of the gaseous form of arteriosclerosis.

T. B., aged sixty years, was well until three years ago, when he began a series of attacks of pain over the heart, which were so intense that during an attack sweat would stand out on his forehead. During the attacks he feared he would die. About this time he began to be troubled by flatulence, not so much during the day as after dinner, when he will feel uneasy and walk about in a nervous state for two or three hours in extreme discomfort. He will then raise odorless gas in explosive quantities and feel completely relieved. Nervousness makes the gas worse, but he has found that on a varied diet it is not worse than when his food is restricted. His chief complaint is of recurring attacks of flatulence after dinner. Physical examination showed obvious arteriosclerosis, slight hypertrophy of the left ventricle, with a ringing second sound, tension high. Patient died a few months later during an attack of angina pectoris.

F. E., aged sixty years, has always been well, but under much anxiety and strain for many years. His present ailment began six or seven years ago, when he suffered from gas coming after meals, especially if he made any exertion, as in walking or undressing, or subjected himself to rapid changes of temperature. The gas collects suddenly and in large amounts, and creates a feeling of oppression in the upper abdomen and in the thorax, at times pressing upon the nerves of the right shoulder. When the gas forms he has to stop until he can raise it, and as soon as he is rid of it he feels absolutely comfortable and can play eighteen holes of golf. His wife notices that anything that makes him nervous increases the frequency and the severity of the attacks. There has been no history of precordial pain.

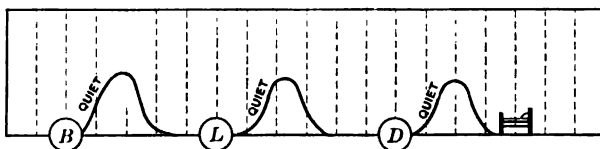
Examination showed a moderate hypertrophy of the left ventricle and an accentuated second aorta sound, faint systolic murmur at the apex; arteries moderately thickened, tension 210 mm. Hg. Test breakfast showed achylia. On the day following the examination patient appeared in great distress, saying that an attack had suddenly come on while in the elevated train, so that it was with great difficulty that he was able to leave the train and take a cab to his office. Examination showed well-marked inflation of the stomach. A tube was passed and a large quantity of gas was expelled under great pressure, with immediate relief to his distress. Within a few days characteristic attacks of angina developed, in one of which he suddenly died.

Pain Type.—Recurring epigastric pain due to arteriosclerosis is described under the term of “angina abdominalis.” The pathogenesis of the pain is often obscure. By some the pain is attributed to hyperesthesia of the sympathetic plexus that overlies the abdominal aorta, while by others the pain is ascribed to a distal localization from painful distention of the aortic ring or ascending portion of the thoracic aorta.

The generally accepted view is that epigastric pain is analogous to the pain of intermittent claudication and is due to a diminished blood supply from vascular colic (*Gefäss Koliken* of Nothnagel) of the affected artery.

For the analogy to be complete one must imagine an increased activity in the gastric wall itself, during increased peristalsis following eating. This reason is probably sufficient to explain the occurrence of pain after hearty meals, but does not explain the connection between pain and exercise requisite to produce it, unless we surmise that exercise raises blood pressure, producing vasoconstriction in the splanchnic

FIG. 122



Pain chart of a patient with angina abdominalis, showing the gradual onset of distress increasing toward the height of gastric digestion, then slowly subsiding even though the patient rests quietly after eating.

area, and narrows the already sclerotic bloodvessels to produce a comparative ischemia of the stomach wall. Pal¹ regards vascular crises to be due to arterial spasm producing cerebral cardiac or abdominal symptoms as well as the paroxysmal phenomenon known as internal claudication. According to Pal a rise of blood pressure precedes and accompanies the crisis of pain, and in a few of his cases spasm or contraction of the retinal arteries could be demonstrated.

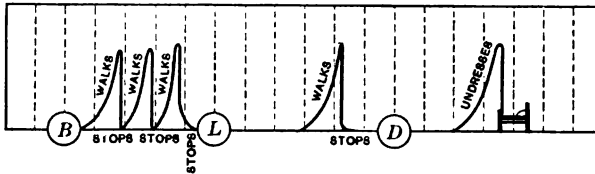
The group of arteriosclerotic patients presenting epigastric pain as a prominent feature may be subdivided into three clinical groups.

1. A dull aching or throbbing pain may be experienced about one hour after eating, which is not due to gas. As a rule the heartier the meal the greater the distress. It is probable that in these cases the narrowed arteries are able to carry sufficient blood to the stomach for its requirements in the quiescent state, but are unable to meet the increased demands of physiological congestion during the digesting state. During active peristalsis the symptoms of ischemia become apparent and the condition is therefore akin to that of intermittent claudication.

¹ *Gefässcrisen*, Leipsic, 1905.

2. There may be no pain after eating unless the patient takes exercise after his meal. This is the most common clinical form of the ailment. If the patient sits quietly after his dinner he is quite comfortable; if he walks he experiences severe pain in the lower thorax and abdomen, often radiating upward to the throat and down the arms so that he will be obliged to stop. After resting a short time, varying from thirty

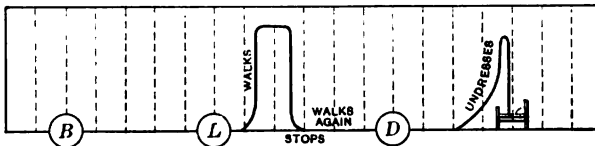
FIG. 123



Pain chart of a patient with angina abdominalis. The pain comes on whenever the patient exercises and is at once relieved by rest.

seconds to a couple of minutes, the pain will disappear and he may then be able to walk without distress for a number of miles. In some cases, however, the symptoms reappear after he has gone two or three blocks, so that he has again to rest until the paroxysm is past. This pain may be relieved by raising gas, although complete relief is not usually so obtained.

FIG. 124



Pain chart of a patient with angina abdominalis. The pain is induced by walking, but after relief from rest does not reappear.

ILLUSTRATIVE CASE.—A characteristic history is as follows:

C. T. P., aged seventy-six years, for the past few years has complained of gas on the stomach whenever he walks after a meal, although he has enjoyed long intervals of freedom. For the past year he has found that if he walks within two hours after a meal he will be seized with a sudden severe pain in the lower thorax and abdomen so that he will have to stand quietly for a minute or two until the paroxysm has passed and then he can walk two or three miles without distress. The heart was apparently normal in size and action although on one occasion there was a slight approach to the bigeminal form of pulse. Well-marked systolic at apex, second aortic accentuated, arteries somewhat thickened, blood pressure 190, systolic murmur heard down the abdominal aorta transmitted to the peripheral arteries. The attacks

decreased in number and severity by appropriate medication. Death from angina occurred within the year.

3. A patient may be suddenly seized by sharp lancinating or crushing paroxysmal pains which recur at short intervals, often every fifteen or twenty minutes, and last but a few moments at a time. Slight icterus has been observed at times, suggesting the possibility of biliary colic. A succession of paroxysmal pains constitutes an attack which may last for several days and be followed by a period of comparative freedom. The attacks are often induced by worry or nervous excitement, and may appear during the night. During the height of pain dyspnea, moderate cyanosis, and Cheyne-Stokes respiration may be present. In a few of the cases a moderate icterus has been observed. These attacks are probably true angina with radiation toward the epigastrium.

Diagnosis.—In all forms of arteriosclerosis there is usually a foreground of general symptoms due to the arterial disease which may be detected by close observation. The physical examination in hospital cases is not of as much importance as one would expect, because evidences of arteriosclerosis are so extremely common in those who have passed into the period of middle age. If it be found that a sudden rise in blood pressure occurs preceding a paroxysm the inference is quite convincing. Steadily high blood pressure while indicating a probable arterial cause for the hypertension does not necessarily indicate that the gastric symptoms in a given case are of arteriosclerotic origin. More important in the diagnosis is the fact that the symptoms are frequently induced or aggravated by exertion, and in the majority of cases no complaint is made if the patient rests quietly after his meal. This same relation between painful symptoms and exercise may occur with perigastric adhesions, and the fact that relief follows the rest of one or two minutes in the sclerotic cases should settle the diagnosis with comparative ease.

Hardly less significant is the effect of medication in arteriosclerotic disorders. There are but few instances of angina abdominalis in which the symptoms are not noticeably relieved by the nitrites or by diuretin.

The gastric analysis in the writer's cases has almost regularly shown achylia to exist, but the number of examinations of the gastric contents is small compared with the number of patients in whom the clinical diagnosis of angina abdominalis has been made, as the writer regards the *presence of arterial disease that gives symptoms a positive contra-indication to the passage of the tube.*

Prognosis.—The prognosis is bad for ultimate recovery, although the duration of the disease may be uncertain. Relief may be expected to some extent at least from treatment, but the possibility of fatal angina at any time renders the outlook distressingly uncertain.

Treatment.—The treatment is that of arteriosclerosis in general, the details of which need not be gone into fully in this connection. Tea, coffee, and tobacco need not be totally eliminated but should be decidedly restricted. Whether or not alcohol is to be totally interdicted is to be decided upon the merits of each individual case. In general, total abstinence is desirable, but in those who are accustomed to the daily use of alcohol with their meals, whisky and water may be permitted at dinner, although the allowance should be small. Alcoholic indulgence, except at meals, should be absolutely forbidden.

Exercise should be taken in moderation and while extreme restriction is generally inadvisable, strenuous exertion, hurrying for trains and rapid running or walking should be avoided. Exercise during the period of pain is contraindicated. Many patients will push on no matter how severe may be their distress, hoping to walk it off. This is never advisable.

The diet should be simple, nutritious, and not too bulky at any one meal. Hearty dinners are often provocative of nocturnal distress, and in some instances the attacks may be minimized by the adoption of a light supper as the last meal of the day in the place of the conventional dinner.

Exercise after meals must be taken sparingly and should cease the moment distress appears, until the paroxysm is past. The bowels should be carefully regulated and a morning aperient draught is frequently of service in diminishing the frequency and severity of the attacks.

Medical Treatment.—We possess in diuretin a remedy which is followed by the most excellent results in the gastric form of arteriosclerosis, and which frequently acts almost as a specific in controlling the attacks.

Diuretin or the double salicylate of theobromine and sodium is best given in aromatic water. In syrup it is apt to deposit, while in powders it is liable to decompose in a short time. The ordinary dose is grains vij-x, three times a day, occasionally pushed to grain xv doses at these intervals, and may be given in the form of the following prescription:

R—Diuretin ʒv
 Aq. cinnamom. ʒiv
 M. Sig.—Teaspoonful in water three times a day after meals.

Or the following prescription:

R—Theobromine sodium salicylat. ʒv ʒij
 Mucilag. acaciæ ʒss
 Aq. menth. pip. ad ʒiv
 M. Sig.—Shake well. Teaspoonful in water three times a day after eating.

The only objection to its use is the costliness of the drug.

Tincture of *strophanthus* in 5-minim doses seems to sustain the effect of diuretin after this drug has been discontinued, and encouraging results often follow its use. The arterial dilators, nitroglycerin, sodium nitrite, and erythrol tetranitrate, are serviceable in reducing blood pressure and in controlling the tendency toward vascular colic. Nitroglycerin by reason of its rapid action is eminently useful to check the paroxysm when acute symptoms appear. The patient should regularly carry with him tablets of grain $\frac{1}{100}$, care being taken that they are not too old, and 1 to 3 of these may be taken at the time of the attack. The action of nitroglycerin seems to be more marked and constant when the patient is taking steady doses of diuretin. The iodides or sodium nitrite may be given (grain $\frac{1}{2}$ -j), preferably in alkaline solution. The action of the latter is slower though more pronounced than that of nitroglycerin. The drug may be well combined with the bromides or with Hoffman's anodyne, as in the following prescription:

R—Sodii nitrit.	grs. viij
Spt. ætheris co.	3ij
Elix. aurantii	3ij
Liq. ammon. acet.	ad 3iv

M. Sig.—3ij every three to four hours in water.

Tablets of erythrol tetranitrate [grain $\frac{1}{2}$ (Merck)] may be given three or four times a day, and are said to exert a sustained action in reducing blood pressure. The writer has, however, been disappointed in this medication and regards it inferior to the other forms of treatment. The iodides constitute the standard form of treatment, but the disadvantage of their prolonged use in producing iodism is obvious. The syrup of hydriodic acid (Gardner's) may be substituted, although somewhat less efficient. If iodides are given they must be well diluted, and preferably combined with a carminative such as tincture of capicum or ginger, as in the following prescription:

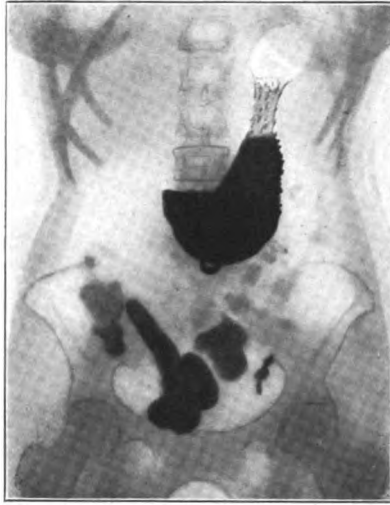
R—Kali lodi.	3ss
Fld. ginger (soluble)	3ss
Syrupus simpl.	3ss
Aq. cinnamomi	ad 3iv

M. Sig.—3j contains $7\frac{1}{2}$ grains potassium iodide.

Sajodin in $7\frac{1}{2}$ -grain doses may be given in capsules four times a day, and has frequently been of service.

PLATE XV

Fig. 1



Lane's Kink. Plate taken six hours after the first bismuth meal and five minutes after the second bismuth meal, showing the advance column of bismuth in the terminal portion of the ileum. The recently filled stomach is moderately atonic. (Radiologist, Dr. Leaming.)

Fig. 2



Lane's Kink. Plate taken six hours after bismuth meal, showing advance of column of bismuth in the lower ileum. (Radiologist, Dr. Leaming.)

DUODENOJEJUNAL OBSTRUCTION BY KINKS AND ARTERIO-MESENTERIC CONSTRICTION

Pain and regurgitation of duodenal contents occur whenever there is obstruction in the duodenum below the ampulla. These symptoms may result from cicatricial contraction following ulceration, although such a low-lying ulceration is rare. More commonly moderate chronic obstruction is found in the terminal portion of the duodenum, or at the duodenojejunal junction, and may be caused in one of two ways: (1) by duodenojejunal kinks, and (2) by arteriomesenteric constriction. The symptoms produced by both of these conditions are practically identical, and there seems to be no way at the present time of differentiating between them. Moreover, the mechanical factors that contribute to the obstruction are in great measure the same, so that the same surgical treatment is required in both instances.

Duodenojejunal Kinks.—The tendency for viscera to drop from gravity, results in the formation of thickened bands in the mesentery which represent nature's effort to reinforce weak points. Unfortunately, this conservative process is not uniform nor sufficient in some instances to overcome the tendency to traction by gravitation, so that kinks are produced that lead to more or less obstruction.

The most important vulnerable point is at the junction of the duodenum with the jejunum, a point which is usually held up by a peritoneal band fixing the end of the duodenum, while the jejunum is unsupported at its commencement. Normally a distinct angulation at this point does not exist, but occasionally the jejunum is dragged down vertically, producing a sharp kink. The drag or traction power is usually due to the dropping of other viscera, especially the lower ileum, which becomes loaded should there be any delay in the passage of its contents into the cecum, so that the heavy end coils of the ileum drag upon the mesentery and pull down the whole of the small intestine. In consequence the jejunum is dragged vertically downward and a sharp angulation on the duodenojejunal junction is produced. The primary cause for the angulation, therefore, is ileal stasis, presumably due to ileal kinks. Lane's ileal kink is the term applied to a pathological bend in the terminal six inches of the ileum, producing a sharp angulation which is complicated by thickening of the mesentery and adhesions of the two arms of the angulation to each other, eventuating in a definite intestinal stasis.

The mechanical result of the kink is a dilatation of the proximal portion of the duodenum, which ultimately becomes congested and occasionally even ulcerated. According to Jordan,¹ who has given us

¹ British Med. Jour., June 1, 1912.

the greatest and most convincing description of the condition studied radiographically, the duodenum may writhe vigorously for as long as nine hours to force the bismuth emulsion past the point of angulation. The stomach also contracts in these cases as though struggling against abnormal obstruction, although the bismuth meal passes freely through the pylorus.

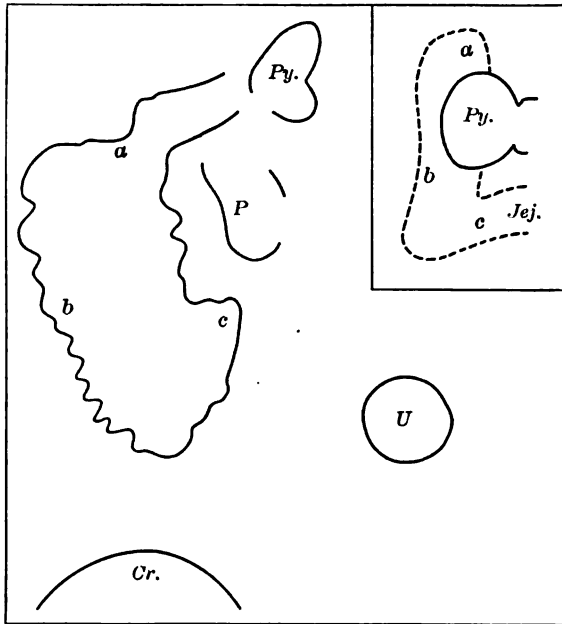
Symptoms.—Pain and vomiting are commonly observed, the pain being due to the efforts of the duodenum to empty itself during the period of its greatest distention. Vomiting may occur at this time and presents the characteristics in some instances of duodenal regurgitation, the ejecta being copious, bile-stained, and occasionally containing pancreatic ferments. The symptoms are not permanent but gradually pass away when the patient lies down and the downward traction on the jejunum ceases. A few days' rest in bed will usually be sufficient for the time being to effect temporary cure; the discomfort, however, reappears after the reassumption of the upward posture, especially toward the close of the day. Pain during the late afternoon or evening is regularly worse than that in the morning. Intermissions in the severity of the complaint, or even complete absence from all discomfort for a number of days may occur during the course of the malady. Jordan (*loc. cit.*) has reported an interesting case with radiographic findings so instructive and convincing that it may be well to cite it in this connection.

A single woman, aged thirty-two years, had suffered from dyspepsia troubles for one and a half years, and during this time had lost nearly 14 pounds in weight. There were loss of appetite and nausea, but no vomiting. She complained of pain in the region of the gall-bladder, saying that she felt there was something distended or gorged in that region which would burst if she tapped it. The distended feeling was also felt in the corresponding point of the back. The attacks of pain were intermittent and were oftentimes absent for days. The patient's general condition was characteristic of intestinal stasis. She was sick and depressed; her hands and feet were cold, her complexion muddy in contrast to her former clear skin; the breasts showed the nodular condition of chronic mastitis; the stomach was elongated and dropped, but normal in other respects. Bismuth emulsion passed through the pylorus. The duodenum was found to be much elongated and dilated to more than double its normal diameter, except the first part, which seemed to be the seat of cicatricial contraction.

"The duodenum was undergoing powerful peristaltic contractions, amounting to strong writhing movements; this continued for seven or eight minutes without a particle of bismuth being able to pass through into the jejunum. The writhing duodenum was observed

and demonstrated to two colleagues without difficulty, for the duodenum was completely isolated in the fluorescent screen picture, the rest of the bismuth being contained in the cardiac end of the stomach. At the end of seven or eight minutes, with a very powerful duodenal contraction, a large mass of bismuth emulsion was sent

FIG. 125



Fluorescent screen tracing (reduced in size) of the duodenum and pylorus in a woman, aged thirty-two years, with the typical symptoms and signs of intestinal stasis. Taken on the couch. The duodenum is half as long again and more than double the width of a normal duodenum. For seven or eight minutes the duodenum was observed undergoing vigorous "writhing" contractions, in a vain endeavor to force its contents into the jejunum through the kink at the duodenojejunal junction. After seven or eight minutes a very powerful contraction of the duodenum forced a large mass of bismuth emulsion through suddenly into the jejunum, and the bismuth forthwith began to course rapidly through the coils of the small intestine. The small figure in the right-hand corner represents a normal (though somewhat distended) duodenum drawn to the same scale for comparison. The distended duodenum of this patient is shown *in situ* in Fig. 2, and an actual skiagram of the duodenum in Fig. 3. The primary cause of the duodenojejunal kinking is shown in Fig. 4. The patient's main symptom was pain in the region of her duodenum, "like something distended which would burst if it were tapped upon either in front or behind." a, b, c, first, second, and third parts of the duodenum; Py., pylorus; P, pyloric portion of stomach; Jej., jejunum; U, umbilicus; Cr., crest of ilium.

suddenly through the duodenojejunal junction and forthwith began to course rapidly through the coils of the jejunum. The subsequent examinations afforded a perfect illustration of the fact that the duodenojejunal kinking is secondary to stasis at the lower end of the ileum. After a night's rest, fifteen hours after the bismuth meal, the stomach

and duodenum no longer contained any bismuth; the greater part of it was found to be in the lower coils of the ileum in the pelvis. The cecum also occupied the pelvis and contained a quantity of bismuth, while a small amount was already present in the ascending and transverse colon. Twenty-seven hours after the bismuth meal there was still some bismuth in the lower end of the ileum, and the most advanced portion had reached the sigmoid, thus the sojourn of the bismuth in the small intestine was more than three times the normal."

Diagnosis.—The diagnosis is suggested by the dependence of the pain on the upright posture and its relief by rest, as well as by coexisting phenomena of intestinal stasis, indicated in the history of Jordan's patient.

Treatment.—The treatment, properly speaking, should be surgical. The question of the propriety of ileosigmoidal anastomosis as advocated by Lane is still *sub judice*, with perhaps an increasing reluctance on the part of conservative surgeons lightly to undertake the task. It would be proper, however, if obstructive duodenal kinking appeared as a constant feature, either to perform gastrojejunostomy with duodenal inclusion, or to straighten out the kink by fixing the first portion of the jejunum in position by suture. The operation which seems to be growing in favor, though still *sub judice*, is exsection of the cecum, ascending, and a portion of the transverse colon.

Chronic Arteriomesenteric Constriction.—The root of the mesentery containing the superior mesenteric bloodvessels passes from behind forward to lie across the duodenum, compressing it against the vertebral column. A normal pressure is always maintained at this point, but so slight as to be easily overcome by duodenal contractions, so that no actual obstructing compression results.

In some instances, however, causes which produce mechanically a downward traction on the root of the mesentery may result in actual duodenal compression.

The relationship between arteriomesenteric constriction and gastrop-tosis has been considered under the latter heading.

Under "acute dilatation" will be found a full description of the relationship between this disease and duodenal constriction by a taut mesentery.

Aside from these cases there is a clinical group characterized by recurring attacks of pain and duodenal regurgitation, in which arterio-mesenteric constriction can be demonstrated by surgical exploration.

The patient usually gives a long antecedent history of obstinate constipation. After a variable period of time there occur attacks of pain, nausea, and vomiting, the vomited matters being profuse and containing bile and other constituents of duodenal secretions.

After the attack has lasted a few hours the symptoms subside until, after a time, the complaint repeats itself.

Exploration in these cases has shown a movable cecum dropping downward over the brim of the pelvis, and dragging upon a mesentery that is abnormally short. Both factors seem to be essential, the downward displaced cecum and the abnormally short mesentery.

Bloodgood, of Baltimore, has called attention to these cases, and proved the pathogenical mechanism of the complaint.

The majority of the patients in whom these conditions have been found have been treated surgically by exsection of the ascending and a portion of the transverse colon, so that fecal stasis would not drag upon the shortened mesentery.

Duodenal Regurgitation Due to Excessive Amount of Fats in the Diet.

—Bassler, of New York, has described a series of cases giving the same clinical history, which he attributes to duodenal regurgitation, occasioned by an excessive diet of fats. According to this writer the pain may be severe enough to incapacitate the patient for the time being, coming suddenly, often disappearing abruptly but quite independent of the meals. The fasting stomach usually contains duodenal secretions, bile fats, and fatty acids, the composite liquids occasionally attaining the quantity of from 50 to 75 c.c. The test breakfast is abundant, amounting to 900 c.c. in one of Bassler's cases, is bile-tinged, and shows a floating layer of fat globules. In the filtrate the pancreatic fermentation may be demonstrated. Reactions for hydrochloric acid depend upon the degree of neutralization by the alkaline secretions of the duodenum. If the gastric contents be removed at the time of pain, reactions and appearances are observed similar to those of the test breakfast. Recovery promptly follows the enforcement of the fat-free diet. Whether Bassler's cases constitute a clinical entity remains to be seen. The writer has no experience in such advanced cases as have been described by Bassler, although he has encountered instances of a milder type.

Differentiation from duodenal kinks and arteriomesenteric constriction has not seemed to him to be possible.

GASTRIC CRISIS OF TABES DORSALIS

Ever since Delamarre¹ in 1866 studied the relationship of the gastric manifestation of tabes to the disease itself and Charcot² in 1881 more

¹ Des Troubles Gastriques dans l'Ataxie Locomotrice, Thèse de Paris, 1866.

² Leçons sur les Maladies du Système Nerveux, Paris, 1800, t. ii, p. 19.

carefully analyzed the clinical features of the paroxysm, the gastric crises of tabes have been classed among the most striking phenomena of clinical medicine.

When they occur in patients in whom the existence of tabes is plainly demonstrable they concern chiefly the neurologist, but not uncommonly they occur in those in whom other evidences of tabes are utterly lacking, even after most painstaking examination, until several years after the beginning of the attacks. In such cases their occurrence is a matter of extreme importance to all clinicians, and, therefore, they demand attention when the symptoms of gastric disorders are being considered.

The frequency of this symptom-complex is variously stated by different authorities. Roestel¹ concludes that as many as 30 per cent. of all cases of tabes show gastric crisis at some time during the course of the disease, while others place the figure as low as 10 per cent. or even 6 per cent.

When a history of luetic infection may be obtained it will usually be found that the attacks first make their appearance on an average of six to seven years after the date of infection, although this is by no means invariable. Cases have been recorded in which the crisis developed within two or three years after the initial infection. They are most frequently first observed in the preataxic stage of the disease and may occur several years before changes in pupillary reactions and patellar reflexes can be detected.

Symptoms.—One of the most characteristic features of gastric crisis in tabes is the suddenness with which the transition is made from a condition of apparent comfort to one of extreme agony. Prodromas are almost unknown. The patient at any time and without the slightest warning is suddenly seized with violent paroxysms of abdominal pain, usually beginning in the lower abdomen or groin and radiating to the epigastrium, in which it may remain fixed throughout the attack. Sometimes the patient feels as though painful constriction was being made about the waist. The pain radiates to the precordium, sometimes to the intrascapular region, and less frequently down the arms.

The onset of the pain is accompanied or soon followed by vomiting, first of stomach contents and then of clear liquid, containing mucus, bile, or even blood. The vomiting and retching seem to become more violent after the stomach contents are expelled and are often uncontrollable. Vomiting may be preceded by choking sensations and loud eructations of gas and hiccoughs. Throughout the attack gas may

¹ Beiträge zur Pathologie und Therapy, bei Crises Gastrique bei Tabes Dorsalis, Inaug. Dissert., Berlin, 1893.

be expelled from the bowel and exceptionally there may be abdominal distention. Constipation is the rule, but diarrhea when present is severe and often very exhausting.

In severe attacks of considerable duration the condition of the patient becomes distressing. The pain is most agonizing and terrifying and the patient thrashes about the bed and assumes most unusual attitudes in his efforts to find relief. As the loss of so much fluid by vomiting continues with the impossibility of retaining anything in the stomach, emaciation is rapid. The skin is pale, cold, and clammy, the pulse rapid, growing weaker as the attack continues. The temperature is almost always normal or subnormal. The abdomen is usually flat and retracted and tender everywhere, but most of all in the epigastrium. Succussion sounds are absent. Areas of cutaneous hyperesthesia and anesthesia may often be found.

The duration of a single attack varies from ten or twelve hours to two weeks or even longer, the average being from five to seven days. Most striking is the abruptness with which the majority of cases terminate. Not only does the pain completely disappear, but in cases of moderate duration a patient who a few hours before was suffering violent pain and vomiting incessantly, calls for food which he eats with relish and seems perfectly able to digest. When the attack has persisted for several days cessation of the pain is often followed by a long sleep, after which the digestive functions seem perfectly restored.

In not all cases, however, are the attacks followed by so prompt a recovery. In many the return to health is gradual, and there may be a longer or a shorter period of impairment of the digestive powers. Exceptionally the attacks may terminate fatally from cardiac exhaustion or from long-continued and profuse diarrhea.

The extreme variations in the gastric crises is exhibited in the same individuals in different attacks as well as in different individuals. The most constant features are the sudden onset, abrupt termination, pain, and vomiting. Very rarely is vomiting absent and almost as rare are those attacks in which there is vomiting without pain but with the other manifestations of the disorder. In duration and the relative prominence of different groups of symptoms a single attack will present the widest departure from the average. It is, therefore, impossible to make any clinical classification of the different types of gastric crises, and equally impossible in a given case to make any prognosis as to the duration and frequency of the attacks.

Variability is a characteristic also of the course followed by the crises. In some cases attacks of two or three days' duration may return with a semblance of regularity every month or six weeks, or they may recur in this manner for several years and then may become entirely

irregular. Irregularity is far more common in the duration of different attacks in the same individual. Sometimes an interval of several years frequently may be enjoyed. In a few cases the attacks have been known to disappear permanently after two or three paroxysms. This, however, is rare. It is much more common for them to increase in frequency and severity as the spinal disease progresses.

Diagnosis.—Gastric Analysis.—Von Noorden¹ concluded after a series of examinations of the gastric contents of tabetic patients made before, during, and after the crises, that no characteristic changes took place in the gastric secretion. He found very variable degrees of acidity which bore no apparent relation to the paroxysm.

The work of observers in more recent years tends to support this view. Friedenwald and Leitz² after studying the gastric contents of the series of 42 cases of tabes in which gastric crises occurred, all males between the ages of twenty-nine and sixty-four, found the results as follows:

In 35 patients from whom the gastric contents were obtained during crises 6 showed local acidity, 13 showed hyperacidity, 10 showed hypoacidity, 6 showed variable acidity. In 36 patients from whom the gastric contents were obtained between the crises, 14 showed normal acidity, 12 showed hyperacidity, 10 showed hypoacidity. Of the entire series—42 patients—only 4 showed hypersecretion.

Treatment.—During the attack the chief indication of treatment is to relieve the patient as far as possible of his pain and distress, and some form of sedative or narcotic treatment is almost always necessary. The simplest and least objectionable drugs should at first be tried. The writer has found as much relief by the combined use of veronal and trional in small repeated doses as from any one of the other forms of treatment. These drugs are best given in combination, in doses of $2\frac{1}{2}$ grains each, repeated every two to four hours. A calmative effect is observed almost from the start and a prolonged and restful sleep is produced when the paroxysm ceases. Antipyrine in 15-grain doses may be given every four to six hours, preferably by rectum, but it should be discontinued if depressant symptoms occur. Cerium oxalate in 5-grain doses every few hours is of good report, but has proved invariably disappointing in the writer's experience. If the paroxysm be not controlled by the foregoing medication it may be necessary to resort to codeia, morphine, or hyoscin. Codeia should first be used as the most free from objection; morphine should be withheld as long as possible. Extreme care must be employed in the use of these

¹ Pathologie der Gastrischen Crisen Charité Annalen, 1890.

² Maryland Medical Journal, July, 1912.

narcotics, as the pain may create a temporary tolerance which ceases when the paroxysm suddenly subsides, leaving the patient unpleasantly under the effects of the narcotic. Hyoscin must be given with extreme caution, as the writer has seen alarming symptoms from doses generally considered to be safe. It is advisable, therefore, not to give the drug in larger doses than grain $\frac{1}{240}$, which, however, may be repeated at such intervals as may be necessary.

The epidural injections of cocaine have been recommended by Ewald. The writer has no experience with this form of treatment. Counter-irritation to the epigastrium and cupping of the spine may apparently relieve for the time being.

There are no specific rules for diet. Food may be given in any form or in any way that seems advisable. There is no reason for starving the patient, nor does food produce an increase in the distress. After the attack is over the Wassermann test should be made, and if positive, active antisyphilitic treatment should be employed, especially by the intravenous injections of salvarsan.

Förster of Breslau is an advocate of resection of the posterior nerve roots in severe recurring crises and has reported¹ that of 25 cases undergoing such an operation 23 recovered, 2 died. Of the 23 surviving patients the results were as follows:

No relapse	13 cases
Relapse	7 cases
No result	2 cases

These statistics are quite encouraging, especially as improvements in technique may be followed by even more brilliant results.

VISCERAL CRISES IN ERYTHEMAS

There is a very important group of cases, in which, to the obvious cutaneous manifestations of the erythemas, including purpura, urticaria, and angioneurotic edema, there are added various visceral manifestations, most commonly colic, with nausea and vomiting and diarrhea, and often hemorrhages from the mucous membranes. The pathology of these conditions is little understood, but it is presumed that the visceral symptoms are the result of changes in corresponding organs, dependent on the instability of the vasomotor mechanism. This underlying condition has been termed "vasomotor ataxia" by S. Solis Cohen.²

¹ Lancet, July 8, 1911.

² Amer. Jour. Med. Sci., February, 1894, cvii, 145.

The classification of the cases which make up this most remarkable group, according to the type of skin lesion present in each case, is extremely difficult, because of the wide variety and difference in the cutaneous manifestations. Not only may these be strikingly different in cases presenting very similar visual symptoms, but also in the same individual they may assume a different form in the different attacks. They are, however, essentially due to vascular changes.

Osler,¹ to whom we are very largely indebted for bringing this group of cases to the notice they deserve, classes them as follows:

1. Cases of pure angioneurotic edema.
2. Cases in which the associated skin lesions is urticaria.
3. Cases of Henoch's purpura, with arthritis, erythema or purpura, and colic.
4. Cases with erythema multiforme, with or without edema, and most frequently with more or less redness or purpura.
5. Cases showing recurring colic, and nothing else, often for years before the appearance of any skin lesion.

S. Solis Cohen² considers Henoch's purpura apart from the others, calling attention to its severity and "probable dependence on definite toxins, if not on specific infections." He states, however, that there is probably some fundamental relationship between them.

In observations of the actual gross changes found in the organs, we are indebted to those surgeons who have operated upon patients during a visceral crisis. The operative findings at these times vary somewhat. Most of the cases show a small amount of free fluid (100 to 200 c.c.) in the peritoneal cavity, which may be clear, turbid, or distinctly bloody. The intestinal wall may, for the space of several inches, be extremely swollen and edematous, and perhaps dark red; the last portion of the ileum seems to be a favorite site for such a swelling. The intestinal peritoneum is usually congested, and may be studded throughout with petechiæ and larger ecchymotic areas. One operator³ reported that most of the intestine was pale and in spasmodic contraction. It is usually found collapsed. Either petechiæ and ecchymoses or the swelling of the intestinal wall may be entirely absent. If the case is operated on during the interval between attacks—sometimes because the gall-bladder has been thought to be the cause of the trouble—the abdominal organs may appear quite normal.

Etiology.—Members of certain families are predisposed to this condition, as might be expected, for heredity has a notable influence on the

¹ Johns Hopkins Hosp. Bull., vol. xv, p. 260.

² New York Med. Jour., 1910, vol. xci, p. 366.

³ H. M. Silver, Amer. Jour. Surg., May, 1909.

erythemas. Ensor¹ cites a family of eighty of whom thirty-three had been affected with angioneurotic edema, and in the well-known case mentioned by Osler this disease was traced through five successive generations of the same family.

As the disorder is prone to recurrences throughout life, the previous history of the patient may show that he has suffered repeatedly from disturbances which are common evidences of vasomotor instability as transient skin eruptions with or without vague attacks of arthritis, asthma, hay fever, or repeated unexplained hemorrhages; also from hysteria, hyperidrosis, drug idiosyncrasies, vertigo, migraine, pseudo-angina, transient hemiopia, and other visual disturbances, and intermittent polyuria. It is not uncommonly present in sufferers from pulmonary tuberculosis, angina pectoris, valvular heart disease, chorea, and epilepsy. Raynaud's disease in some of its forms may be considered an associated condition in which the vasomotor disturbance is manifested by local constriction rather than by dilatation.

A host of influences, many of them seemingly trivial, may serve as the exciting cause of an attack; among these are reflex excitation of any kind, cold, emotion, and toxins formed in the body, especially in the gastro-intestinal tract; for example, strawberries or shell-fish, which act as poisons when eaten by certain individuals.

Symptoms.—The type of patient in whom these conditions are found is an important part of the picture. There are usually the evidences of marked instability of the vasomotor system: dermatographia, peculiar mottlings of the skin, rapidity and extreme variability in rate of the heart action, which is easily disturbed; or irregularity, palpitation, or intermittent tachycardia. Functional murmurs may be found. Besides this there have been described in many cases a widening of the commissure of the eyelids, tremulousness of the lids on light closure, dilatation of the pupils, which react but show wide oscillation.

During an attack the character of the skin eruption may be variable in the extreme. There may be purpura of various degrees—sometimes resembling measles or erythema, with urticaria, or nodules with a dark centre. The only feature in any way approaching constancy is the distribution, which is most often over the extensor surfaces of hips, knees, ankles, and elbows. Exceptionally the only discoverable area of eruption may have other less expected locations. There may be only limited evidences of angioneurotic edema, and in the difficult cases embraced in class five of Osler's classification no skin lesion may be visible.

Recurring attacks of colic are a far more constant feature than are

¹ Guy's Hospital Reports, vol. lviii, p. 111.

many of the cutaneous manifestations. These come on at variable intervals of a month or more, sometimes permanently disappearing, but usually exhibiting a marked tendency to persistent recurrence. The onset may be accompanied by a chill. The pain is violent, often beginning in the epigastrium and becoming diffuse over the entire abdomen; in the other cases it may be confined to the lower half of the abdomen, or it may be very indefinite in location. Radiation is not common, but cases have been known to very closely simulate biliary or renal colic. Nausea, vomiting of stomach contents, clear, greenish, or bloody fluid, and diarrhea with the passage of blood, sometimes following constipation, are fairly constant accompaniments of the colic.

During an attack of colic the patient usually appears extremely ill. The abdomen presents no constant signs, but often there is general rigidity and tenderness over the colon, appendix, or epigastrium, or in all of these locations. Distention is rare, the abdomen often being decidedly retracted. In some cases a mass has been distinctly felt, which subsequent operation has shown to be the swollen portion of intestine as above described. The spleen is moderately enlarged in a fair proportion of the cases. A moderate rise of temperature up to 101° is common. The blood usually shows no changes in coagulation time, a moderate leukocytosis—up to 14,000 (although two cases, showing over 30,000, have been reported)—and a normal differential count. Among the less common but important features of the attacks are albuminuria, with casts and blood, hemorrhages into any mucous membrane or retina, and arthritis.

A very severe form of acute nephritis is a most important if not the most common complication. It is not infrequently the cause of a fatal termination, and may first make its appearance when the skin manifestations are at their height, or not until several days or even weeks after their disappearance. Osler¹ draws attention to the fact that in the most intense form of the nephritis there may be no edema. In his series he has never met with nephritis as a complication in a case of pure angioneurotic edema. Endocarditis, pericarditis, pleurisy, and pneumonia are rare complications.

Intussusception complicated a case reported by Sutherland.² Its importance lies in the similarity of the symptoms of an attack with colic to those of intussusception. Peritonitis resulting from a perforation of the fundus of the stomach is reported by Silbermann³ in a case which showed necrotic foci in stomach and intestinal walls, but without

¹ Amer. Jour. Med. Sci., 1904, cxxvii, 19.

² British Jour. Child. Dis., 1904, i, 26.

³ *Pediatrische Arbeiten, Festschrift Herri Edward Henoch*, 1890, p. 239.

ulceration. There had been recurrent attacks of fever, pains in the knees, purpura, colic, and melena.

While the gastro-intestinal crises are the most frequent of the visceral manifestations, nevertheless crises apparently resulting from disturbances in other organs often occur. Such are aphasias, hemiplegias, monoplegias, swelling of the fauces and pharynx, edema of the glottis, and asthma, which seems to be especially frequent with urticaria. The causative structural changes are thought to resemble those found in the abdominal organs.

Diagnosis.—It must not be forgotten that certain forms of the so-called exudative erythema are frequently secondary to organic disease, such as chronic valvular cardiac disease, hepatic cirrhosis, cholelithiasis, and nephritis. An important step in diagnosis therefore is the exclusion of these organic lesions.

The type of patient in whom the attacks occur, shown both by a careful history and physical examination, has no small influence in deciding the question of the nature of the attack. Attempts to differentiate the attacks from colic with abdominal inflammatory conditions, by means of the symptoms and physical signs alone, are attended with much difficulty on account of the diversity with which the symptoms of the former are grouped. It is always to be borne in mind that the two conditions may coexist.

In acute appendicitis the vomiting at the onset, if present, is more likely to be of stomach contents only and of shorter duration, and bloody stools are much more rare than is the case with the visceral crises. Tenderness is usually more marked and more definitely localized, and rigidity is much more constant.

Intussusception is extremely hard to differentiate, especially in the absence of a cutaneous eruption. A careful inquiry for a history of previous attacks of erythema, and a searching examination for the minutest evidence of the present existence thereof, in all cases presenting paroxysmal abdominal symptoms, will greatly aid in avoiding errors.

Treatment.—At present we know of no means of preventing a recurrence of the attacks in these cases. Camphor and arsenic both have their advocates, but more extended trial is necessary before their use is attended with any certainty of success.

The question of surgical intervention is an important one. While we cannot agree with the view of some surgeons that most cases showing the symptom-complex under discussion are benefited by operation, still serious inflammatory conditions coexist in a sufficient number of cases to make operation by far the lesser danger in cases of great doubt.

Osler emphasizes the importance of rest in bed and a milk diet for a sufficient length of time to guard against the development or progress of the nephritis.

EPIGASTRIC HERNIA

Epigastric hernia may occur in two forms. The first or true hernia occurs whenever, through a cleft in the abdominal wall a peritoneal sac is projected, containing usually omentum, occasionally portions of the small intestine, or even of the wall of the stomach itself. This form of hernia is well known and needs no further description.

A second form of hernia, often unrecognized, is frequently the cause for prolonged dyspepsia, and the description is limited to this variety.

Epigastric hernia, known also as preperitoneal lipoma, consists in the intrusion of a small mass of preperitoneal fat through a minute cleft or deficiency in the aponeurosis of the abdominal wall. The mass varies in size from the head of a pin to a small chestnut, and is composed almost entirely of subperitoneal tissues, although it is possible that a cone-like projection of the peritoneum itself may be drawn into the orifice.

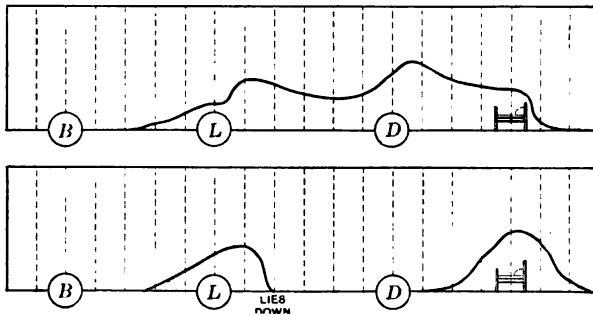
The ordinary situation for the hernia is in the median line between the umbilicus and the xiphoid. So commonly are they found in this situation that the term "hernia of the linea alba" is often employed to designate them. A not infrequent situation is the upper portion of a patulous umbilical ring, which rounded at its lower circumference terminates in the upper portion in a narrow slit, the outline resembling a pear, the lineal cleft representing the short stem. Less frequently hernia appears at the outer edge of either rectus or engages in clefts in the inner layer of the sheath of the rectus muscle. The hernia may be single or multiple, rarely, however, exceeding three or four in number. The disorder is far more common in men than in women.

Etiology.—The exact mode of origin of the hernia is unknown. The cleft may represent congenital deficiency or may possibly result from minor traumatism, although the clinical history rarely gives any intimation of previous injury. The history of a rapid loss of weight preceding the hernial symptoms is so commonly elicited that it would seem as though disappearance of fibrous fat has allowed the entrance of the herniated tissues through a cleft thus previously occluded. It is certain that little masses of subperitoneal fat may penetrate the spaces formed by the fibers of the linea alba, especially around the course of the small vessels and nerves which pass through these spaces. If this little mass continues to grow, the opening through which it passes

is stretched about the pedicle of the hernia so that it exerts a pressure upon it whenever the abdominal wall is rendered tense by muscular effort. To the base of the pedicle the omentum may become adherent.

Symptoms.—The chief symptom is pain localized in the epigastrium, occasionally showing irregular radiations. It may be described as sharp or lancinating, although it is usually of a dull aching character. Although the point of tenderness is extremely minute, the pain may be difficult to locate accurately or may spread over quite an extensive area of the abdomen. Exacerbations in severity are regularly induced or intensified by walking or any physical exertion that demands the free use of the abdominal muscles. The patient may be quite comfortable on waking, but as the day progresses the pain becomes more and more nagging and persistent, gradually waning when the patient rests. Relief by rest is not, however, as instantaneous as in angina abdominalis. The

FIG. 126



Pain chart of a patient with a small epigastric hernia. On the first day the patient did not rest and the pain increased until she laid down at night. On the second day relief came by lying down in the afternoon.

pain may be often intensified after eating during the period of maximum peristalsis. The connection between the motor activity of the stomach and the localized hernial pain is not readily to be explained, especially as internal adhesions do not necessarily constitute a part of the morbid process.

Vomiting is neither infrequent nor especially characteristic. Diarrhea has been noted in several instances, the bowels assuming their normal function when the hernia has been rectified. Irregular attacks of flatulence have also been relieved after the hernia has been removed.

Physical Examination.—The characteristic evidence of the hernia is the presence of a small spot of exquisite tenderness in any of the above-mentioned regions in which hernia commonly occurs. The area of tenderness is usually no larger than the blunt end of a pencil and the line of demarcation is quite distinct. The sensitiveness is intensified

whenever the abdominal wall is put on the stretch, as in attempts to rise from a recumbent to a sitting position. If the patient has been quiet for several days the tenderness may disappear, so that the most favorable time for examination is when the patient is suffering from pain after exercise.

The hernia may or may not be palpable. Small intrusions of fat may be so minute as to totally elude detection. In other cases an extremely tender nodule the size of a small pea, or rarely somewhat larger, may be distinctly felt. The size of the hernia bears no relation to the distress which it produces. Hernias of the size of a chestnut or walnut may be of this type of preperitoneal lipoma, although when they attain this size a true hernia with a peritoneal sac is to be suspected.

Treatment.—The treatment is entirely surgical. A small incision is to be made and the subcutaneous fat in the neighborhood of the painful spot removed down to the aponeurosis. If the hernial fat be detected it may be tied off and the opening closed, care being taken to avoid opening the apex of any possible peritoneal protrusion. In many instances the hernia cannot be found, but a clean dissection down to the aponeurosis and the removal of all subcutaneous fat that immediately overlies the painful spot will result in complete and uneventful recovery.

CHRONIC LEAD POISONING

Lead is a slowly cumulative poison. It makes its entrance into the body chiefly through the alimentary tract, either in water, or on food among those of unclean habits, or as dust which, having entered the upper respiratory tract, is swallowed with saliva. Whether or not it may enter the system directly through the respiratory tract has been much discussed, and can as yet scarcely be considered proved. It is probable that skin absorption, if possible, is not great.

Thus are thousands of men and women who are daily exposed to lead poisoning on account of their work. The variety and number of such occupations are too great for enumeration here. It must not be overlooked, however, that lead is used in the manufacture of dry electric batteries and many kinds of rubber goods, for at first thought these occupations might seem remote from the lead industry. Their importance in increasing because they are becoming more and more extensive.

A complete history, including occupation, is usually sufficient to put the careful examiner on his guard, but even in our present state of civilization there are many other ways in which lead poisoning may occur. Drinking water may become impregnated with a soluble salt of lead; cosmetics, hair dyes, and cheaper grades of thread and dyes for dry

goods may be the source of the poison. Fortunately, there is little chance today of danger on this account from canned foods. Unusual susceptibility to its action exists in certain individuals and families, in those who are regular users of alcohol, and especially in those who have suffered previous attacks.

In the stomach, lead is probably converted into the soluble lead chloride and absorbed as such, but in what form it circulates in the body we do not know. In fatal cases lead is found in many of the organs and tissues, but the lesions produced are far less characteristic than are the resulting symptoms. The discrepancy is so marked that the question of how the poison acts on the tissues in producing symptoms is even now unsettled. Excretion takes place chiefly through the kidneys, bile (lead is consequently found in the feces), and to a less extent in sweat, milk, and saliva.

Symptoms.—Chronic lead poison is usually first shown by a group of general symptoms more or less indefinite, and which are followed after a period of weeks or months by the more characteristic symptoms, colic, neuritis, and severe cerebral disturbances. The first symptoms are increasing loss of weight, loss of appetite, especially for breakfast, a sweetish taste in the mouth, constipation, sometimes alternating with diarrhea, attacks of nausea and vomiting, frontal headache, attacks of weakness and trembling, spots before the eyes, pains in joints and muscles, and a moderate anemia with disproportionate pallor.

The most definite and characteristic symptoms at this stage are the blue line or "lead line" on the gums and the granular basophilia of the red cells. They are usually present at all stages of the poisoning and often when there are no active symptoms.

The blue line is an irregular line of purplish discoloration, very close to and parallel with the edge of the gum, being especially marked about the lower incisors and canines. It occurs on both the inside and outside of the gum. When examined carefully or with a lens it will be seen to be made up of small round dots, situated just beneath the surface. These are deposits of lead sulphide produced by the action of sulphuretted hydrogen liberated from the decomposition around the roots of the teeth on the lead circulating in the gum tissue. Therefore, it is least marked in those whose teeth are kept cleanest, and may be absent when there are no teeth and the gums are atrophied. It persists for months after all symptoms have disappeared.

The blood shows anemia of various grades, the red cells being diminished out of proportion to the diminution of hemoglobin, but red cell counts below two million are rare. Normoblasts are found, but seldom in large numbers. The white cells show nothing characteristic. Granular basophilia of the red cells is practically always present. The

granules take basic stains, and are usually small in size, appearing like fine points. Often, however, the size varies, some being as large as eosinophile granules. Granular basophilia is found in other diseases, but in none of them is it so evidently out of proportion to the degree of anemia, and in this lies a diagnostic sign of the greatest value.

At any time one of the more characteristic symptoms may appear, colic being more often the first. It is apparently due to spasm of the bowel, and is usually preceded by obstinate constipation, and often by premonitory pain of less severity. Sooner or later, and frequently beginning at night, there is a violent outbreak of spasmodic abdominal pain. It may be chiefly umbilical, or epigastric, or diffuse over the entire abdomen. Schmidt¹ states that it is often accompanied by sharp lumbar pain. Vomiting is common. During the paroxysm, the abdominal wall is usually hard and retracted, pressure often giving relief, although at other times the entire abdomen is markedly tender. Between the violent spasms which last a few minutes the patient is in comparative comfort for a short interval. The severity of the attack is variable, some being so agonizing as to cause complete prostration, while others are tolerable. It may last from a few hours, or with remissions may extend over several days. The attacks recur at various intervals if exposure is continued.

The distinguishing characteristic of lead neuritis is its distribution. It eventually produces bilateral wrist drop from paralysis of the extensors of the fingers and wrists. Less frequently and usually later the perinei and extensors of the toes and ankles become involved. The paralysis may become quite general, progressing from the periphery, or more rarely other atypical forms of paralysis may appear at first.

Delirium, coma, convulsions, epileptiform or unilateral, or variable are the most striking cerebral symptoms. They are always dangerous, and sometimes of extremely grave portent. They may be precipitated by unusual indulgence in alcohol, and are said to occur in a larger percentage of negroes than of other patients.

There is no constancy in the order of appearance of the different symptoms. Colic, paralysis, or even coma may be the first indication of illness to the patient or his friends. Other constant but less characteristic symptoms are evidences of arterial and cardiac sclerosis, of interstitial nephritis and ocular symptoms, such as muscular paralyses, hemianopsia, and general and gradual impairment of vision. Of the ocular disturbances some are due to the encephalopathy, others to optic neuritis, and neuritis of the nerves of supply of the external eye muscles.

¹ Pain, its Causation and Diagnostic Significance in Internal Diseases, 1908.

In those who promptly change their occupation or withdraw from the source of exposure, health may return almost completely. If the exposure has continued for some time the resulting damage to kidney, liver, and circulatory system may continue to progress even after the withdrawal of the source of intoxication, and it is from the failure of these organs that death most often occurs in those who escape it in the encephalopathy.

Diagnosis.—A negative history of exposure has little value. Where the source of poisoning is clear, any failure of the general health is to be viewed with suspicion, and when the characteristic neuritis or colic are added to the picture the diagnosis is practically certain. The association of "dry" colic and neuritis is sufficient to make lead poisoning highly probable, and the addition of the blue line makes the diagnosis certain.

When colic is a prominent symptom extreme care must be exercised lest an acute abdominal inflammation demanding prompt surgical treatment be overlooked because of the certainty of the diagnosis of lead poisoning. The two conditions frequently coexist, some authors even going so far as to state that acute appendicitis is especially frequent among sufferers from lead poisoning. The writer once saw a case of acute perforative appendicitis that had been mistaken for lead colic by several men of wide experience. The patient presented all the signs of lead poisoning, and the diagnosis of that condition was unmistakably positive. However, he was sufficiently stout to obscure the local abdominal signs, and as a consequence of the error operation was delayed two days. General suppurative peritonitis supervened, and a fatal termination soon followed.

The blue line when present and distinct is strong corroborative evidence, but if the gums and teeth are in good condition it may be very slight and poorly defined. It is also absent in some early cases which give symptoms. It is almost universally agreed that the most reliable single sign in all stages of the disease is the granular basophilia of the red cells when out of proportion to the grade of anemia. Cabot¹ maintains that lead is the only disease which often produces basophilic stippling in the red cells in the absence of marked anemia, and states also² that he has never known a clear case of plumbism without stippling.

The presence of lead in the urine is of great value in diagnosis, its absence is less helpful, for it is quite probable that it may disappear from the urine intermittently for short periods.

Treatment.—A discussion of the manifold features of the prophylaxis of chronic lead poisoning in the industries is out of place here; it may be summed up as cleanliness of hands and skin, reduction of dust and all

¹ *Differential Diagnosis*, 1911, p. 132.

² *Ibid.*, p. 152.

other possibilities of exposure to a minimum, and strict avoidance of constipation. The free use of milk has the reputation of being a powerful preventive, probably because it not only combines with the hydrochloric acid in the stomach, thus leaving less to form the soluble lead chloride, but also because it forms an insoluble albuminate with the lead.

A person suffering from chronic lead poisoning must be removed from the source of intoxication if possible. The further treatment consists in the elimination of the lead from the system and the treatment of the special symptoms present. Elimination is best promoted by moderate catharsis with magnesium sulphate, together with atropine for its antispasmodic effect when there is severe colic, and enemas, simple or of oil, or containing magnesium sulphate. The magnesium sulphate is the best of the saline cathartics to use for this purpose, because it forms the insoluble lead sulphate with that portion of the lead which is free in the intestine.

Potassium iodide not only increases the elimination in general, but probably also forms the soluble double iodide of lead and potassium, in which form it is excreted, but in the presence of acute symptoms this drug must be used with caution, for it sometimes causes an increase in the toxic manifestations supposed to be due to the sudden liberation in the body of so much lead in soluble form. Not over five or at the most ten grains three times a day should be used, and then not without careful watching for toxic symptoms.

For the colic hot moist abdominal applications, hot baths, and anti-spasmodics are helpful, and finally morphine must be resorted to in many cases, although it checks the process of elimination. The neuritis and encephalopathies are treated on the lines governing the treatment of these conditions when due to other causes.

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